HERTA FLOR

EMPIRICAL EVALUATION OF A DIATHESIS–STRESS MODEL OF CHRONIC BACK PAIN

DISSERTATION
zur Erlangung des akademischen Grades
Doktor der Sozialwissenschaft
in der Fakultät
für Sozial- und Verhaltenswissenschaften
der Eberhard-Karls-Universität Tübingen

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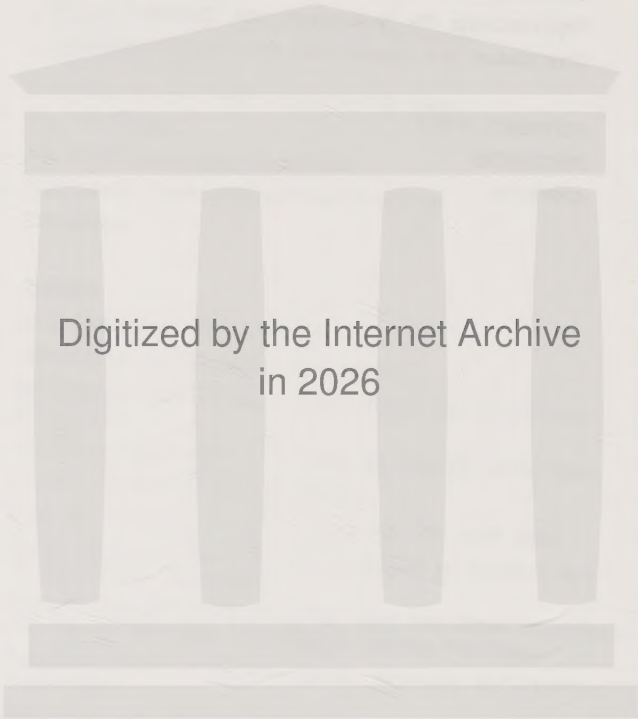

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0. Preface

The topic of this paper is the pathogenesis of chronic back pain (CBP). Basis of the approach used here is a biopsychosocial view of health and disease as proposed by Engel (1977) and Schwartz (cf., 1982). This means that both biological as well as psychosocial variables are thought to be important contributors to the experience of CBP. Theoretical as well as empirical research on the influence and interaction of these factors will be presented.

This paper is organized in seven sections. Section 1 will discuss the scope of the problem CBP and describe its features. Sections 2 and 3 will describe and evaluate somatic and psychosocial models of CBP. In section 4 a comprehensive diathesis-stress model of CBP will be proposed. An experiment that was designed to test the major assumptions of the model will be described in sections 5 (methods) and 6 (results). In section 7 the empirical findings will be discussed and their implications for diagnosis and treatment of CBP will be considered.

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Tübingen, September 1983

1. SCOPE OF THE PROBLEM OF CBP

Back pain is a very common disorder. It affects about 50 - 80 % of the population at some point in their lives (Hirsch, Jonsson & Lewin, 1969; Hult, 1954, 1965; Svensson & Andersson, 1982). It is the third leading cause of physical limitation and disability in the United States with 3.9 % of the population permanently disabled by it (Vital and Health Statistics, 1974). Of the 1.25 million people in the United States who suffer back injuries every year 65,000 patients will be permanently disabled (Beals & Hickman, 1972). In Germany, back pain is usually classified as a rheumatic disorder. Rheumatological problem affect about 10 million Germans (Ciba Geigy, undated). The majority of those suffering from rheumatism are affected by back pain (e.g., Wagenhäuser, 1973). Rheumatism or CBP respectively are the leading cause of premature disability in Germany (Müller & Boos, 1971) and the United States (Haber, 1971). Only 50 % of the patients who have had back pain for more than 6 months will return to work.

CBP reaches its highest prevalence between 40 and 60 years, its peak of onset is between 20 and 30, with men and women about equally affected. Although not life-threatening, CBP is a pervasive disorder leading to much suffering for the patient and his or her family. Its socioeconomic costs are enormous. An estimated 10.6 billion German Marks were spent on rheumatic disorders in 1976 (Ciba-Geigy, undated) and 14.6 billion dollars were spent on CBP in 1978 in the United States (Schaepe, 1982).

Despite of its prevalence, the impact on the individual, and its socioeconomic costs, little is known about its etiology. A range of diagnostic terms is associated with CBP, for example lumbosacral strain, lumbar disc disease, osteoarthritis, sciatica, lumbago, spondylitis, spinal stenosis, and so forth (e.g. Benn & Wood, 1975; Blumberg et al, 1964; Cailliet, 1981; Finneson, 1980; WHO, 1967).

Altough meant to be explanatory these terms are usually merely a description of symptoms because little is known about the etiology of chronic back pain (c.f. Murphy, 1977; Nachemson, 1980). Benn and Wood (1975) note: "Such imprecise diagnoses as muscle strain, ligamentous sprain, or simply a description of symptoms (lumbago) account for 50 - 90 % of the diagnoses" (p. 121).

It is important to note that back pain is a symptom, not a disease. Back pain can be an indicator of malignancies, visceral diseases, vascular disease, and so on (e.g. Cailliet, 1981; Mankin & Adams, 1980). Acute back pain is usually a self-limiting condition, receding even without medical consultation (e.g., Dixon, 1980). Estimates are that 90 % of acute back syndromes improve within 2 months (e.g., Dillane et al., 1966; Horal, 1969; Hult, 1954). The problem, however, is the high recurrence rate. An estimated 90 % of the patients relapse (Hirsch et al., 1969) with each recurrence becoming more severe and long-lasting (Horal, 1969). The term CBP ist commonly used to refer to back pain whose assumed origin is in the spine or the surrounding tissue and is not due to a specific disease process but is thought to be of degenerative, muscular or inflammatory origin and has lasted for more than 6 months. It is characterized by dull, aching, continuous pain that typically affects the lumbar and cervical areas, often spreading to the legs. Restricted motion and feelings of stiffness may accompany it, often in combination with strong paravertebral muscle spasms (e.g., Albrecht, 1975).

Despite the magnitude of the problem chronic back pain and the abundance of literatur on the topic, amazingly little is known about its causes and hence its effective treatment (e.g., Nachemson, 1979, 1982). In the follwing two sections somatic as well as psychological approaches to the etiology of CBP will be described and evaluated.

2. SOMATIC APPROACHES TO THE ETIOLOGY OF CBP

The purpose of this chapter is to give a brief overview of traditional medical models of CBP and evaluate their adequacy. Detailed descriptions of the various clinical pictures presented in this review and detailed discussions of empirical evidence on these approaches can be found in Cailliet, 1981; Evans, 1982; Finneson , 1980; Jayson, 1980; Macnab, 1977, and others.

Numerous etiological factors have been suggested as being of importance for CBP. Back problems have been classified into several categories, according to their assumed pathogenesis (cf. Albrecht, 1975): (a) inflammatory (e.g.,ankylosing spondylitis, nerve root inflammation), (b) degenerative (e.g., disc hernia, spondylosis), (c) structural (e.g., congenital spinal deformities), (d) traumatic (e.g., injury to the spine), (e) muscular, ligamentous (e.g., myalgia). Research on each of these categories (except (a), which is very rare), will be reviewed.

2.1. Degenerative disorders

Degenerative disorders of the spine have been considered to be of primary importance among the causes of CBP. Several specific degenerative mechanisms have been postulated. Although they often appear together, they will be discussed seperately and only the most important features of degenerative disorders of the back will be included. Research evidence on each of these factors will be reviewed in turn.

2.1.1. Degenerative Disc Disease

The most often assumed degenerative mechanism is degenerative disc disease. In most people in the western world structural disintegration of the inververtebral disc (Spondylosis) occurs as a common aging process. Fibrillation of the nucleus pulposus, cartilage degeneration in the endplates leading to disc space narrowing, ruptures of the annulus fibrosus and so forth can be observed. These processes may lead to subsequent affection of the adjacent vertebral bodies and joints and osteophyte formation. If protrusion or prolapse of the disc (i.e. rupture or bulging of the annulus fibrosus or sometimes protrusion of the nucleus pulposus) occur, impingement of the adjacent spinal nerves may ensue, leading to pain radiating down the legs (sciatica), and often

sensory and motor losses. This nerve root involvement, however, seems to occur only in a small percentage of the population (cf. Loeser, 1980). A consequence of disc degeneration may also be narrowing of the spinal canal (stenosis). Although these processes have been well described and can be demonstrated radiologically (cf. Finneson, 1980), it remains largely unclear how they may produce pain (cf. Nachemson, 1982).

Schmorl and Junghanns (1932) showed in their classic autopsy studies of 4253 spines that degenerative changes of the spine are present in 50 % of the population by the end of the fourth decade, 70 % at the end of the fifth decade, and 90 % at the age of 70. Radiological studies show similar numbers of degenerative changes in the general population (e.g., Hult, 1954). Thus, disc degeneration seems to be a common aging process that occurs in everyone - in western industrialized societies, that is. Fahrni (1975) noted that this degeneration does not occur in non-industrialized societies. Comparing CBP patients with healthy controls, Moral (1969), Hult (1954), and Hussar and Guller (1956) found equally high incidence of degenerative changes in the x-rays of healthy controls and chronic back pain patients. In a similar, more recent survey Magora & Schwartz (1980) found that single disc degeneration occurs even more often in people who have never had back pain.

None of the control-group studies found any relationship between radiologically documented changes of the spine and back pain. In addition, x-ray based diagnoses are very subjective and unreliable. Nachemson (1982) notes: "One can draw almost any subjective interpretation from looking at a series of x-rays. There are no consistent radiological findings associated with injury of the back that one can lead to the etiological cause of back pain" (p. 60). Dixon (1980) emphasizes that disc prolapse is probably the most overdiagnosed disorder of the back and concludes: "Indeed, the whole myth of the 'slipped disc' is one of the deep-rooted weeds that have sprung up in this area. Discs do not 'slip' in the way which laymen and not a few doctors imagine ..." (p. xi). Wyke (1980) suggests that only 5 % of the back pain patients really suffer from prolapsed disc. Wyke's (1980) estimate is particularly important since many treatments are based on the assumption of disc herniation or prolapse. This is especially true for back surgery which may only - if at all - be indicated for a very

small percentage of CBP patients if disc prolapse led to nerve impingement (cf. Loeser, 1980) - but is performed on about 200,000 Americans anually (cf. Pheasant, 1977).

2.1.2. Osteoarthritis

Another related degenerative process is osteoarthritis (i.e.,degenerative disease of the spinal joint cartilage affecting primarily the facetal or posterior intervertebral joints). This degenerative process involves narrowing of the joint space due to cartilage loss and the formation of osteophytes - especially at the vertebral bodies, with spur formation, ridging and bridging of the vertebrae.

There degenerative changes are, however, as common in healthy controls as they are in patients with back pain (cf. Magora and Schwartz, 1976). Such structural changes have rather been found related to age, overweight, scoliosis or hyperlordosis, and may consequently be viewed as due to wear and tear. These changes may also occur secondary to disc degeneration (e.g., Hirsch, 1966) but cannot be considered as the sole etiological factor for back pain since spinal osteoarthritis is often asymptomatic (cf. Mankin & Adams, 1980).

2.1.3. Spondylolisthesis

Another degenerative process (which may also be a congenital anomaly), spondylolisthesis (i.e. forward shift of one vertebra onto another due to joint derangement) has also been thought to be a cause of back pain. Although some authors found an increased incidence of CBP when spondylolisthesis was present (e.g., Horal, 1969; Hult, 1954; Torgerson & Dotter, 1976), others observed no such relationship (e.g. Rowe, 1969; Splithoff, 1953). Since no prospective studies were conducted it remains unclear to what extent the degenerative changes may be causes or consequences of CBP.

Although this overview of assumed degenerative processes in CBP is not exhaustive, I have mentioned the major factors that are proposed in the literature.In sum, no relationship between degenerative changes of the spine and back pain has been demonstrated consistently. Very severe changes may at the worst aggravate existing back pain.

2.2. Structural (Congenital) Anomalities

Congenital (or acquired) structural anomalities of the spine such as spina bifida occulta, hyperlordosis, kyphosis, scoliosis, transitional vertebrae, have often been viewed as important etiological factors in CBP (e.g., Cailliet, 1981; Stinchfield & Sinton, 1955). Pain may be induced by injury or is assumed to be due to spinal instability, ligamentous or muscular strain etc. But several studies demonstrated that those anomalities are unrelated to CBP (e.g. Horal, 1969; Hult, 1954; Magora & Schwartz,1978, 1980; Torgerson & Dotter, 1976).

2.3. Muscular and Ligamentous Dysfunctions

Muscular and ligamentous dysfunctions have also been implicated in the development of back pain. Muscle spasm and atrophy are often found in CBP patients and are viewed as a result of pain, spinal derangement or postural faults (e.g.,Evans , 1982; Finneson, 1980; Wyke, 1980). Fibrositis, described as an inflammation of paravertebral muscle tissue has often been considered as a pathological entity but there seems to be no pathoanatomical basis for this assumption (cf. Fassbender, 1973; Nachemson, 1982).

Ligamentous strain and ruptures have also been implicated as possible causal factors for CBP (e.g., Howes and Isdale, 1971; McNab, 1977). However, the research evidence does not support their role (e.g., Nachemson, 1982).

Only few authors have addressed the possibility of muscular dysfunction as an important pathogenetic factor in CBP. Kraus (1965), Kraus & Raab (1961) and DeVries (1966) assumed underexercised muscles would spasm during work, and would become fatigued, both processes resulting in back pain. These assumptions are, however, speculative, with few data to support the hypotheses. Studies that examined the muscle tension levels of back pain patients are equivocal. Some authors found differences between healthy controls and CBP patients in resting muscle tension levels in various body positions (e.g., Grabel, 1973; Hoyt et al., 1981; Kravitz et al., 1981); others did not find differences (e.g., Basmajian, 1976; Collins et al., 1982). DeVries (1968) demonstrated increase in muscle tension levels that coincided with pain increase in patients with CBP during prolonged standing. These discrepancies may be

largely due to methodological problems. Small numbers of subjects, uncertainties about normal tension levels, unclarified or divergent selection criteria for the patients under study, varying methods for EMG measurement (e.g. needle versus surface electrodes), differences in the site of measurement and insufficiently controlled experimental designs do preclude firm conclusions from these studies (see also sections 3 and 4).

In a different approach, Fassbender (1973, 1975) took biopsies from the trapezius muscle in patients with upper back pain. He observed muscular atrophy and degeneration of muscle fibers in the hypertensive trapezius muscle. He assumed constant muscular hypertension and subsequent ischemia and anoxia to be the cause for these changes in the muscular substrate, and postulated constant muscle spasm as an etiological factor in CBP. Cailliet (1981) also suggested that various kinds of stress may lead to muscular hypertension and pain without, however, providing any data. Since most kinds of physical therapy are geared toward alleviating muscle spasm , it would be important to demonstrate its existence and the underlying dysfunctional processes. Although the current research failed to demonstrate a clearcut relationship between muscle spasm and back pain, it did not refute it either. More research using improved methodolgy may clarify these issues.

2.4. Traumatic Injury to the Back and Occupational Factors

Traumatic injury to the back may result in acute back pain. This may be due to many microtraumata as well as a single more severe injury. But noticeable injury to the back has not been found of major importance in the pathogenesis of CBP. Only 6 to 28 % of the back pain patients name a specific precipitating event like injury when asked (e.g. Hult, 1954; Horal, 1969; Rowe, 1969). Parachutists, for example, who are exposed to a great deal of spinal trauma, do not have a higher incidence of back pain (Murray-Leslie, 1977).

Certain occupations, especially those that require long driving, heavy manual labor or frequent bending and lifting have been related to an increased incidence of back pain (cf. Anderson, 1971, 1980; Schaepe, 1982). Several studies, however, found no relationships between occupation and back pain (cf. Hult, 1954; Magora, 1970 a, b; 1972; 1973 a; Rowe, 1969). Long sitting (driving) seems to have some relationship to

the incidence of CBP (e.g., Kelsey and Hardy, 1975) but the evidence is far from conclusive. Magora (1970) reported that the incidence of back pain correlated best with how stressful workers perceived their job to be and not how physically demanding it really was. Stressful work as perceived by the patient, problems at the workplace and in the family may be more closely related to the incidence of back pain than "objective factors" (Dehlin et al., 1976; Magora, 1970 a, b; 1972, 1973b; Westrin et al., 1972). Similarly, Nerell and Waghlund (1977) found that the incidence of CBP doubled when psychological work stress (dissatisfaction with job, alienation from work) was present. Schaepe (1982) suggests that work stress may lead to nervous tension, over-recruitment of muscles and subsequently to CBP. This relationship will be discussed in more detail in sections 3 and 4. A caveat is necessary, however. All those studies are cross-sectional and correlational and thus the direction of the causation cannot be determined.

Overall, the negative conclusions of Wood and Badley (1980) seem to be warranted: "We can find little to refute the suspicion that most published work has sought only for evidence consistent with the author's preconceived notions ----striking inconsistencies tend to be ignored, neglecting the fact that that differential morbidity rates between occupational groups can present useful opportunities for falsification of hypotheses" (p. 42).

2.5. Summary

Wood and Badley's (1980) conclusions can be extended to most of the research on the pathogenesis of CBP. If one tries to delineate the possible causes of CBP one is thrown back on speculation with little empirical basis. Neither degenerative, nor structural, traumatic or occupational factors seem to have any clearcut relationship to CBP. The research on muscular involvement in CBP is also equivocal. Moreover, a large range of symptoms and syndromes is assembled under the 'umbrella' of CBP - with a wide range of terminologies and descriptions, mostly without sound empirical basis. This is especially troubling, since many of the treatment approaches offered are based on these questionable assumptions regarding the pathophysiology of the chronic back pain problem. Nachemson (1979) summarizes the situation accordingly: "Having been enganged in research in this field for nearly 25 years and having been

clinically engaged for nearly the same period of time, and as a member and scientific adviser to several international back associations, I can only state that the true cause of low back pain is unknown" (p. 143) ..."since the cause is unknown there is only symptomatic treatment available" (p. 145). Medical treatment of CBP has therefore been of little effectiveness (for a review of somatic approaches to the treatment of CBP see Flor and Turk, in press).

Various methodological problems make it hard to evaluate the research on somatic models of CBP in general. What is needed, are studies that carefully define and measure patient characteristics, use adequate control groups and determine the base rate of various disorders in the population before citing abnormal physical findings as a cause of CBP. It is important to note that most of the research adheres to a back pain patient "uniformity myth" (Kiesler, 1966). Back pain patients differ from each other in physical, psychological and social variables, which may all interact in very specific ways. It would seem important to identify the specific factors that determine the back problem in a patient and identify their interaction. A major problem of the medical research is that for a long time it adhered to a purely somatosensory model of pain - thus disregarding its multidimensionality (cf. Melzack, 1973; see also section 3.). This led to an almost complete disregard of psychosocial antecedents and consequences of CBP. Only recently has the potential importance of psychosocial factors as determinants of CBP besides organic factors been noted (cf. Sternbach et al., 1973 a, Westrin et al., 1972). Fordyce (1978) has especially emphasized that learning of pain behaviors may take place, turning an acute into a chronic pain problem. Disregard of these factors and lack of efficacy of the traditional medical treatment approaches have forced many patients into a "low back loser" (Sternbach et al., 1973b) career with changing physicians, growing medical records, disability, depression, and overutilization of the health care system without appropriate relief. Increasingly, health care professionals are recognizing the importance of psychological and social factors in pain problems and they have started to incorporate them into their conceptualizations of the pathogenesis and the treatment of CBP. The following section will review the theoretical rationale as well as empirical evidence for the role of psychosocial factors in CBP.

3. Psychological Approaches to the Etiology of Chronic Back Pain

The lack of a definitive organic etiology for CBP and the ineffectiveness of many of the medical treatments have elicited a multidisciplinary attitude towards the study and treatment of CBP. CBP is increasingly viewed as a psychophysiological and psychosocial problem resulting from the interaction of physical, psychological and social variables all of which are considered important contributors to the pathogenesis of CBP. There is also a growing recognition of the impact of back pain not only on the patient's body but on his or her job, family and other social relationships, and the need to consider these factors in explaining the maintenance of CBP as well as to address them in treatment.

Two developments were of special importance for this new view of pain -- (a) the proposal of the gate-control model of pain by Melzack and Wall (1965; see also Melzack, 1973; Melzack & Dennis, 1978; Melzack & Loeser, 1978), (b) the rise of the disciplines of health psychology and behavioral medicine (cf. Melamed & Siegel, 1980; Pomerleau & Brady, 1980; Prokop & Bradley, 1980) - - that provided the methodology to assess the role of psychosocial factors in the etiology of CBP and provided innovative treatment approaches.

Traditional (somatic) approaches to pain defined pain as a purely sensory event with the pain experience assumed to be directly proportional to the peripheral damage. The many instances of pain without discernible peripheral damage (cf. Sternbach, 1974) as well as the clinical reports of absence of pain experience in spite of severe damage (e.g., Beecher, 1959), however, showed that this is too simplistic a theory. Factors like the meaning of the situation, previous experience, attentional and attitudinal processes and so forth, are implicated as important determinants of the pain experience. Melzack and Wall (1965) tried to account for these observations in their multidimensional model of pain - the gate control theory. The theory describes the pain experience as a result of the complex interaction of sensory-discriminative, affective-motivational and cognitive-evaluative components. Their assumed physiological substrate are multiple afferent and efferent, ascending and descending neuronal impulses modulating the noxious input. Although anatomical and physiological aspects of the theory remain uncertain (cf. Nathan, 1976; Wall, 1978) this model has stimulated a host of new re-

search and treatment approaches. It provided a theoretical physiological basis for the effect of psychosocial factors on pain and for the mechanisms of action of psychological treatments.

On the empirical side, the research of Magora (1970 a, b; 1973 b) as well as Westrin et al. (1972) suggested that psychosocial variables may be more closely related to CBP than many of the assumed somatic factors. Psychologists have therefore increasingly been involved in research on the etiology and treatment of chronic pain (cf. Sternbach, 1978).

Most psychological models do not try to explain the development of acute back pain but rather try to account for the transition from acute to chronic back pain. The first research efforts have only fairly recently been made and the models are therefore not yet very elaborate nor is the empirical evidence to assess their adequacy sufficient. They do, however, point out potientially important contributors to the pain experience that have not yet received sufficient attention.

3.1. Psychodynamic Approaches

The first to propose and describe psychological factors in CBP were psychoanalytically oriented authors in the 1950's and 1960's. Their hypotheses are largely based on uncontrolled case studies and their psychoanalytic interpretation with little empirical evidence available.

Theoretically, psychoanalysts have described back pain as conversion neurosis, depressive equivalent, or hypochondrical reaction (cf. Adler, 1981 ; Blumer, 1982). Many of these assumptions are based on Freud's (1952) description of pain as a conversion neurosis resulting from a compromise between the fulfilment of a forbidden wish and its punishment. Reich (1933) exphasized the relationship between anal-retentive character traits and chronic muscular hypertension as often observed in CBP.

Engel (1959) has described the "pain prone" patient as an individual for whom psychic conflict is important in the pathogenesis of pain states with or without an organic basis. He emphasizes the symbolic meaning of pain that gives it the property of an important means to regulate the psychic economy. The pain-prone individual is described as

someone with a background of guilt who will often have depressive features and who uses pain as a means of self-punishment to attenuate guilt (via somatization), who experienced a history of defeat and intolerance of success, whose family history shows the important role of aggression in relationships. The pain syndrome may appear when the unconscious need to suffer cannot be satisfied otherwise, when real, imagined or anticipated loss occur, or it may be evoked by aggressive or sexual impulses. The choice of the pain site is explained via unconsious identification with a love object. Engel (1959) did not provide empirical data to demonstrate the adequacy of his assumptions.

Blumer and Heilbronn (1982) expanded on Engel's (1959) work. They suggest that pain of uncertain origin is a specific form of depressive mood disorder with characteristic clinical features. They describe those pain-prone patients as displaying continuous pain, with a hypochondriacal preoccupation, a desire for a surgical solution, inactivity, lack of initiative, fatigue, and insomnia. Additional features are described as denial of difficulty in interpersonal relationships with idealization of family relations, strong dependency needs, denial of hostile-aggressive impulses combined with an eagerness for social approval, but inability to enjoy social life, leisure time and sex.A history of "workaholism " has been noted with intolerance of success, as well as a family history of abuse, and often a relative with disability, sometimes CBP. They describe the pain-prone patient as shifting from 'solid citizen' to an invalid who uses his or her pain problem as a culprit for dependency needs. They suggest that treatment should focus on antidepressant medication in combination with operant treatment (e.g., Fordyce, 1976 a, b).

Another line of theoretical reasoning directly addresses the choice of the back as the site of pain and assumes that specific situations and conflicts lead to involvement of the back muscles.

Grace and Graham (1952), in their specific attitudes theory, describe the typical back pain patient as having phantasies of running away to avoid stressful situations. The inability to actualize this response tendency presumably manifests itself as chronic tension and subsequently as pain.

Alexander (1951) similarly assumed an inhibition of aggressive im-

pulses leading to a simultaneous activation of antagonistic muscles and subsequent muscular hypertension and long-lasting pain states .

Dorpat and Holmes (1962) formulated a related theoretical concept. In their opinion individuals with a "back attitude" (i.e. those with a tendency to perform motor acticity involving the back muscles when experiencing difficult social and life adjustments) tend to display a muscular fight-flight response. This response tendency is thought to be inhibited by their feelings of guilt and fear of injury. They further hypothesize that this inhibited fight-flight response leads to sustained muscular contraction, subsequent ischemia, oxygen depletion and the release of pain-eliciting substances. These processes lead in their opinion to a vicious pain-tension cycle. Herrmann and Schonecke (1981) described a similar pathogenetic model of chronic extraarticular rheumatism.

Several uncontrolled studies tend to support these hypotheses. Janus (1975, 1978) studied the trapezius EMGs of patients with upper back pain during psychoanalytic interviews and sessions. He found EMG increases when aggressive impulses or anxiety-provoking conflicts were addressed, and decreases during the verbalization of fantasies, especially flight fantasies.

Holmes and Wolff (1952) reported that neurotic patients with back pain showed muscular hyperactivity during movement or during interviews provoking conflict and hostile feelings. Dorpat and Holmes (1962) found increased EMG activity in the upper and lower back as well as in the hamstring muscles in 65 back pain patients as compared to 10 healthy controls. This increased activity occurred when the patients were asked to perform simple exercise or during psychoanalytic interviews that addressed personal conflicts, expecially feelings of hostility, guilt and frustration. They report, however, no statistical data. Dorpat and Holmes (1962), as well as Janus (1975, 1978), interpret their findings as indicative of the patients being constantly on guard, whith quick mobilization of the body musculature leading to sustained muscular hyperfunction and subsequent pain. They suggest this generalized muscular hyperfunction to be pathogenetic in CBP. Blumer and his collegues (1982; Blumer & Heilbronn, 1982) present some inital evidence for their assumption of chronic pain as a form of depressive disorder. Support for their

assumption comes also from the fact that a substantial number of chronic (back) pain patients are depressed (cf., Turner, 1982). It is, however, not clear whether depression is an antecedent, a consequence or a syndrome occurring simultaneously with CBP. The studies presented above often suffer from lack of adequate control groups, from lack of statistical analysis, and from methodological problems in EMG measurement.

The psychoanalytic models described above are usually rather vague, not well formulated nor comprehensive (for an exception see Blumer & Heilbronn, 1982). They especially do not address why the generalized muscular contraction assumed in some models (cf., Dorpat & Holmes, 1962) should lead to a specific back pain syndrome but not other forms of muscular pain. They fail to consider the base rates of individuals with conflicts but no back pain, as well as those with back pain and no identifiable conflicts and also fail to incorporate the possible role of other somatic factors in CBP. In addition, all studies were conducted with patients who already suffered from CBP. Thus it cannot be determined whether abnormal muscular acticity is a cause or a consequence of CBP. Prospective studies would be needed to address this question. Although the approaches described above still lack theoretical sophistication and empirical validation, they nevertheless raise interesting issues and suggest new lines of research.

3.2. Family Systems Theory

While psychoanalytic models view intrapsychic conflicts as a major source of the development of CBP, family systems models emphasize the role of social interaction in the pathogenesis of CBP as an additional factor. Various authors used different approaches to explain the development and function of bodily symptoms from a family systems viewpoint (cf., Roy, 1982). Although these theories do not usually address CBP specifically, they are applicable to this syndrome as well. Family systems theory views the family as a system of relationships in which the functioning of each member is interdependent (e.g., Jackson, 1957). Symptoms are believed to be an expression of a familial conflict or to serve a role in stabilizing the family system. Weakland (1977) argues that the large body of evidence on the influence of emotional stress on

bodily functioning is proof for the importance of interactional and familial variables for the disease process, since emotional stress stems largely from communicative interaction.

Waring (1977) explicitly refers to chronic pain in his model of "significant other specificity" as a mechanism of selection of the symptom and of the symptom bearer. The basic idea is that symptoms of the patient fulfill the emotional needs of other family members (e.g., projective identification, conflict avoidance through the symptom). Symptoms may appear at random (for example, acute pain) and may turn into a chronic pain problem if they fulfill the object-related needs of a family member via a scapegoating mechanism or projection. Those needs may lead to reinforcement of the symptom and the subsequent "sick-role homeostasis" may stabilize the family system. Grolnick (1972) suggested that rigidity in family interaction may lead to the development of psychosomatic illness, while Meissner (1966) suggested that somatic decompensation of an emotional crisis in an emotionally overinvolved family may lead to the development of a psychosomatic syndrome.

Minuchin and his collegues (1975) proposed a comprehensive model of the development of psychosomatic illness in children that has also been applied to chronic pain (cf., Liebman et al., 1976). They suggest that a certain physiological vulnerability, combined with four transactional characteristics of the family (enmeshment, overprotectiveness, rigidity, and lack of conflict resolution) and the use of the child for conflict avoidance, are important preconditions for the development of a chronic pain problem. They suggest that the choice of the symptom may be determined by the family history. Its immediate precipitation may be caused by a threat to the family homeostasis and the maintenance of the symptom is explained by the new stabilty which the symptom gives to the family system.

There is little research on the influence of the family on the development of chronic pain or CBP. A special problem posed by family systems theory is that it adheres to a model of circular causality that is hard to test with conventional research methods based on a model of linear causality. Several authors report evidence that the patients, siblings and relatives of patients with chronic pain problems have substantially higher numbers of pain complaints than controls, thus supporting the notion

of pain "running in families" (e.g., Blumer & Heilbronn, 1982; Gentry et al., 1973).

Two studies (Mohamed et al., 1978; Shanfield et al., 1979) reported high incidence of similar pain complaints in spouses of chronic pain patients and their families. The latter studies provide evidence for Waring's (1977) assumption of significant other specificity.

In sum, both the psychoanalytic and the family systems models offer interesting ideas about the development of chronic pain states, and they take into consideration psychosocial factors which medical models tend to neglect. To date these approaches are not yet well-developed and usually only address one specific psychological aspect of chronic pain problems. While the psychoanalytic approaches delineate specific types of emotional (intrapsychic) conflicts as causes of CBP, the family systems models discuss the influence of interpersonal conflicts in the pathogenesis of CBP. They fail, however, to describe the translation process from a psychological problem into a physical symptom, and thus do not integrate physical and psychosocial factors in their model. Empirical evidence for these models is also still sparse. Most data are based on subjective, often retrospective reports. Further development of the models and more refined and precise research is clearly needed.

3.3. The Respondent Model

The respondent as well as the operant approach (see 3.4.) to CBP are behavioral models of chronic pain that do not make specific assumtions about back pain, but believe the processes they delineate to be effective in all kinds of chronic pain. The respondent model as described by Gentry and Bernal (1977) suggests that classical conditioning of pain and tension may occur in an acute pain state, due to some form of physical damage leading to a pain-tension cycle. Pain is viewed as an antecedent as well as a reaction to muscular hypertension. Avoidance of movement may be used to reduce pain, leading to increased immobility which may induce still more tension and pain. With time, more and more situations may elicit pain and anxiety. Depression and dependence on medication may follow, intensifying the pain-tension cycle still more. Similarly, Caldwell and Chase (1977) assumed that once an acute pain problem exists, conditioned fear of movement may develop, inducing an avoidance of activity and leading to immobilization. This may result

in muscular atrophy and increasing disability.

Although there are some preliminary data, the empirical evidence for those assumptions is not satisfactory. Several studies examined the relationship between muscular hypertension and back pain. Specifically, they questioned whether back pain patients exhibit higher resting tension levels of the back muscles than do normal controls. Grabel (1973) found a significant difference in resting EMG values of CBP patients and healthy controls in a prone position. Since his methodolgy of EMG measurement is questionable (he used the number of peaks exceeding 10 microvolts), the data are not conclusive. Kravitz and his collegues (1981) observed elevated EMG levels in CBP patients during differential relaxation of the back muscles; this difference did,however, not reach statistical significance. In various other positions they could not find a difference in EMG levels. Hoyt and his collegues (1981) noted differences in EMG levels between CBP patients and healthy controls, but only while the patients were standing, not while sitting. Collins and her collegues (1982), however, could not find any differences between patients with low back pain and healthy controls when measuring their lumbar EMGs in different body positions. On the contrary, they report lower EMG levels in the CBP patients in some positions (e.g., forward bending). Basmajian (1976, 1978) noted similar observations without, however, providing clearcut evidence for these assumptions. Conclusions from all these studies are hampered by methodological prolems. Often only one or few body positions were employed, and the methodology of the EMG measurements varied (e.g.,use of needle versus surface electrodes, and bi- versus unilateral measurements), as well as the duration of the measurements (Collins et al.,(1982) for example, used only 3 x 12 second periods). Often no clear diagnostic criteria were used to describe the patient samples, and the sample characteristics varied from study to study.

3.4. The Operant Approach

The operant model of CBP as prosposed by Fordyce (1976 a, b, 1978 , Fordyce & Steger, 1980) distinguishes between private pain experience and observable and quantifyable "pain behaviors". Only the latter are assumed to be amenable to behavioral treatment and modification.

The model proposes that acute pain may come under the control of external contingencies of reinforcement and thus develop into a chronic pain pain problem. Pain behaviors (e.g., complaining, taking medication, inactivity) may be directly reinforced, for example by attention of the spouse and health care providers, by rest, or by reduction in the pain level via analgesic medication. Pain behaviors may also be maintained by avoidance of undesirable activities such as unwanted sexual activity or work. In addition,"well behaviors" (e.g., working) may not be as reinforcing as pain behaviors, and the more rewarding pain behaviors may thus be maintained.

The model has not been directly tested although the efficacy of operant treatment offers some indirect support. Fordyce and his collegues (1968, 1973), and, more systematically, Cairns and Pasino (1977) and Doleys et al. (1982), demonstrated that pain behaviors (specifically inactivity) can be decreased and well behaviors (activity) increased by verbal reinforcement with or without feedback, and by setting exercise quota. Block et al. (198o) showed that pain patients reported differential levels of pain in an experimental situation depending on whether they were observed by their spouses or by ward clerks. Pain patients with nonsolicitous spouses reported more pain when a neutral observer was present. When solicitous spouses were present, the pain patients reported more pain than with the neutral observer. The spouses were usually more solicitious the longer the pain problem had existed. The study suggests that spouses can be discriminative stimuli for the display of pain behaviors by pain patients.

More research is needed to demonstrate the validity of the model, for example by measuring the amount of operant reinforcement in the patient's natural environment and its relationship to the pain behaviors displayed. The systematic manipulation of well and pain behaviors in acute and chronic pain patients, as well as in experimental analogue studies, and the documentation of histories of reinforcement are also needed. The value of the operant model lies in the impetus it gave to behavioral approaches to pain management. The model fails, however, to consider the interaction of somatic, psychological and social factors in chronic pain, focusing on the latter only.

3.5. Social Modeling and Cognitive-Behavioral Assumptions

Several authors have emphasized the role of observational learning or modeling in the acquisition of pain problems. Specifically the "choice of the pain site" has been explained by modeling factors. Craig (1978), for example, describes how social modeling processes may inhibit or disinhibit old reactions to pain, or instigate new reactions to pain. Factors of potential influence may, for example, be cultural habits and views (e.g., Weisenberg, 1977; Zborowski, 1969) or early socialization experiences (e.g., Melzack, 1973), as well as later social (especially familial) interaction (e.g., Roy, 1982). Craig and his collegues (e.g., 1971, 1975 a, b, 1977) demonstrated that modeling may influence intensity ratings and tolerance of experimental nociceptive stimulation.

Meichenbaum and Turk (1976) emphasized the importance of cognitive processes in coping with pain. Maladaptive evaluations of the pain experience (e.g., "catastrophizing") may lead to feelings of helplessness and a sense of loss of control. Thus an acute pain problem may turn into a chronic pain syndrome. Inactivity, depression, and disability may follow. The efficacy of cognitive-behavioral interventions for chronic pain, as well as the vast laboratory research on the influence of cognitive factors on experimental pain, support this idea (e.g., Turk et al., 1983). Lefebvre (1981), for instance, demonstrated cognitive distortions in CBP patients. Indirect evidence comes also from a study by Flor et al. (1983) where improvement in CBP patients treated with EMG biofeedback also resulted in considerable reduction in thoughts expressing helplessness and loss of control in relation to the back problem. There is still a lack of empirical research testing important aspects of the cognitive-behavioral and the observational learning models. At present, then, these assumptions must at best be viewed as suggestive.

3.6. Summary

Psychological models of CBP have addressed psychosocial variables that have been neglected by somatic approaches to CBP. They have raised important issues and generated a host of new treatment approaches to CBP (for a review of psychological treatments of CBP, see Turk and

Flor, in press). Like the medical models, they suffer from a lack of controlled research. Preliminary data are, however, encouraging and suggest the importance of psychosocial variables in CBP. A problem with those models is that they usually focus on only one aspect of the psychological functioning of the individual - intrapsychic conflicts, familial interaction, classical conditioning of pain, tension and anxiety , operant conditioning, modeling factors or cognitive processes - without trying to incorporate them into a comprehensive model, and without considering their possible interaction. They also generally fail to discuss the translation of psychological symptoms into physical pathology, i.e. they do not explore the concurrent psychological and physiological processes in CBP. This is partially so because the models attempt only to explain the change from acute to chronic pain behavior, irrespective of the initial pathology. The operant, respondent and cognitive-behavioral models focus only on this aspect, namely the autonomy of CBP from its original pathophysiology. The other models (psychoanalytic, family systems thoery, social modeling) describe the psychological mechanisms leading to the development of CBP, but fail to sufficiently delineate important aspects of its pathophysiology and psychophysiology. Besides the neglect of the somatic or psycho-somatic angle of the porblem, they also do not sufficiently discuss the social factors involved in CBP. Although family systems theory to some extent addresses social interaction as causative in CBP, it limits its theory to familial interaction, neglecting the potential importance of occupational stress (cf., Westrin et al., 1972) and stress in other social contexts (e.g., dealing with institutions). The operant model also only partially addresses these factors (e.g., by discussing display of pain behaviors as a means to avoid disliked work). Thus none of the models fully addresses the entire spectrum of variables that may be important contributors to the CBP experience. A more comprehensive preliminary model of CBP subsuming many of these propositions under a common conceptual basis will be presented in the following chapter.

4. A Diathesis-Stress Model of CBP

4.1. Theoretical Background

The diathesis-stress model proposed here specifically adresses the development of CBP but may be applicable to other pain syndromes especially musculoskeletal, as well (e.g., myo-fascial pain dysfunction and temporomandibular joint dysfunction syndromes; see Moss et al., 1982). The basis of the model is a biopsychosocial view of health and disease (cf., Engel, 1977; Schwartz,1982), as noted before. A multifactorial or "organismic" view (cf., Schwartz, 1982) of CBP is adopted. This approach to health and disease has been described by Schwartz (1982) as "...the belief that specific diseases (constellations of symptoms) represent the complex interaction of specific environment stresses (including germs) and the organism in question (including its genetic and experiential history) and that biological and behavioral stresses always interact with each other to produce particular constellations of signs and symptoms in particular individuals." (p. 1042; underlined words in italics in the original). The preliminary model described here only partially fulfills these claims: It does adhere to a multifactorial view of pain (cf., Melzack & Wall, 1965; Turk et al., 1983) in that it does not completely neglect the basic social and somatic variables involved. Its primary focus, however, is on the psychophysiology of CBP. This focus is primarily due to the competence of the author in the psychophysiological field, but reflects also the lack of research efforts and evidence in the other fields.

The diathesis-stress model, as described on the following pages, assumes CBP to result from an interaction of potentially stressful environmental events, with a predisposing organic or psychological condition, or diathesis. Davison and Neale (1982) conceptualize diathesis as referring "most precisely to a constitutional predisposition toward illness but the term may be extended to any tendency or inclination a person may have to respond in a particular way to an environmental stress" (Davison & Neale, 1982, p. 64).

Stress is viewed in a transactional model as proposed by Lazarus and his coworkers (Lazarus, 1966; Lazarus & Cohen, 1977; Lazarus & Launier, 1978; Roskies & Lazarus, 1980). The model suggests that a

stress experience may result from the interaction of the person's appraisal of environmental demands and his or her perception of the availability of coping responses. A perceived discrepancy between the demands and the individual's resources will result in an experience of threat, leading to a somatic stress response. The experience of stress is believed to result more from the way an individual perceives and interprets events than from the mere presence of "stressful events" (as suggested by the life event research, cf. Dohrenwend & Dohrenwend, 1974). Lazarus and his collegues (cf., Lazarus, 1966, 1975, 1977; Lazarus et al., 1957; Speisman et al., 1964) demonstrated the important role of cognitive processes in the stress experience and the biological stress reaction.

This model of stress coincides with the view of reciprocal determinism of behavior (cf., Bandura, 1978) and a cognitive behavioral perspective (cf., Turk et al., 1983) that assume a continuous interaction between a person's thoughts and feelings, his or her behaviors, and environmental stimuli. Individuals do not only react to the environment but actively construe their world according to the attitudes, beliefs, and interpretations they bring to a situation, and they act upon the environment according to their idiosyncratic perception of events. This interpretation and subsequent action will also change the environment and elicit further reactions. The stress an individual experiences may be viewed as a result of these interactions.

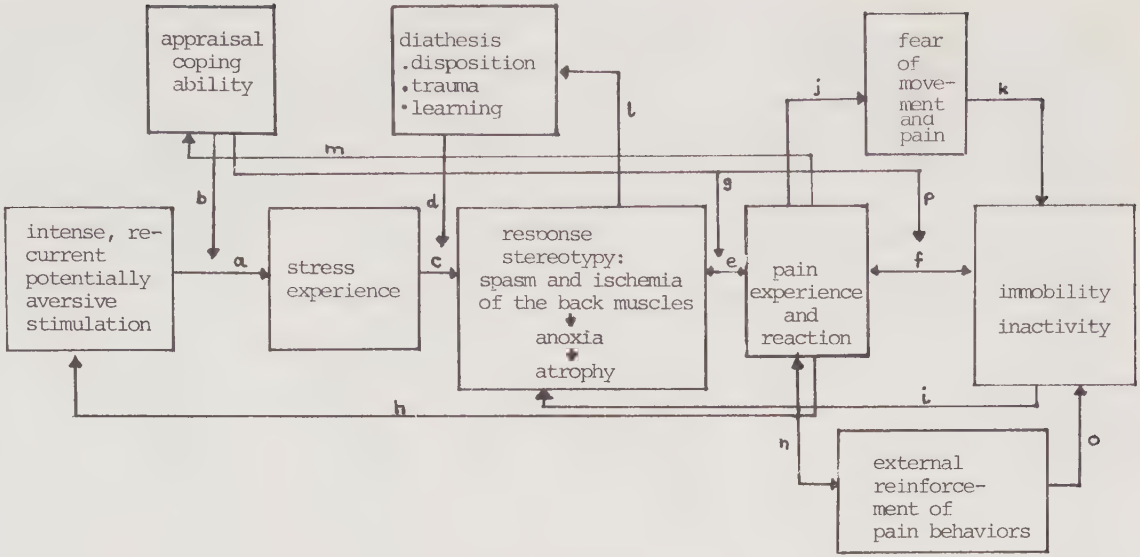
Founded on these concepts and the research presented in the previous chapters (especially the psychoanalytic and respondent approaches, as well as the somatic approach that focusses on muscular deficiencies), a diathesis-stress model of CBP will be outlined.

4.2. Description of the Model

Table 1 presents the key features of the model. Potentially aversive stimulation which is very intense or prolonged (recurrent), and which an individidual cannot adequately cope with, is thought to lead to an increase in muscular tension as a natural reaction in the fight-flight response to stress (cf., Cannon, 1929; see links a, b, c in figure 1). In an unfavorably disposed back, an individual response stereotypy (cf., Lacey & Lacey, 1958; Malmo et al., 1950) may develop, resulting in an extreme and sustained muscular hyperreaction, specifically of the back

FIGURE 1

DIATHESIS STRESS MODEL OF CHRONIC BACK PAIN



muscles (links c, d). Strong emotions stemming, for example, from close interpersonal situations, usually involving family members or fellow workers, are believed to be especially potent stress reactions with concomitant physiological manifestations. The diathesis (link d) may be a congenital disposition for vulnerability of the back muscles, or it may result from previous injury to the back, underexercised muscles, or learning processes, for example. Learning may have occurred in the form of observation of a back pain response from relatives, certain habitual body positions (e.g., due to certain socially approved ways to sit, stand, walk...)leading to strained muscles, or display of certain emotional reactions typically involving mobilization of the back muscles (stimulus-response specificity, cf., Dorpat & Holmes, 1962; Schwartz et al., 1982; Wenger & Cullen, 1958).

This muscular hyperreaction of the back may be mediated by the gamma-motoric (extrapyramidal motoric) system that is closely related to the limbic system, and therefore especially susceptible to emotional influences (cf., Andreasi, 198o; Struppler, 1975). The increase in muscular tension may lead to ischemia and subsequent reflex muscle spasm in the affected area. Ischemia may as well be induced primarily by the mobilization of sympathetic arousal, leading to vasoconstriction and subsequent reflex muscle spasm at the affected site (cf., Struppler, 1975).Both of these processes usually interact when a stress response is activated. Long-lasting ischemia is thought to lead to oxygen depletion (anoxia) in the fatiguing muscle and the release of pain-eliciting substances (e.g., bradykinin) as part of a homeostatic mechanism (cf., Dorpat & Holmes, 1962; see link e in **figure 1.**) Pain then serves as a signal to pay attention to the back and terminate the pain. In some cases the appropriate action - e.g., removal or attenuation of the stress, muscle relaxation - will be taken, depending on the availability of coping resources and the appraisal of the pain signal (link g in figure 1).Often, however, no appropriate action will be taken and a longlasting, recurrent pain problem may develop.

The pain experience can act as a new stressor (links e and h) increasing the stress experience and tension, thus initiating a vicious tension-pain-tension cycle. In addition, pain tends to be experienced as a sign to rest and discontinue activity (link f) in the absence of alternative coping strategies (link p), which may be an appropriate

action in the case of acute externally-inflicted pain but is often inappropriate in CBP. Longlasting inactivity and immobilization of a body part may lead to increased muscle spasm and worsening of the pain problem (links f, i).

Additionally, fear of movement can become conditioned, and avoidance of the fear through cessation of activity may develop (links j,k), since movement of spastic muscles leads to a direct, momentary increase in pain. Immobility may again increase the fear to move (link k), leading to more spasm, pain and stress, and to a vicious cycle that may soon become independent of external stimulation. In addition, external stressors may exert still more influence now, since the pain and tension may increase the vulnerability of the back and the back muscles (link l). Andersson et al. (1974), for example, demonstrated that an increase in muscle tension in the back increases the risk for injury beyond that of muscle spasm described thus far. The tension is directly proportional to the pressure exerted on the disc, causing it, eventually to bulge or rupture (cf., Andersson et al., 1977). Thus the diathesis-stress relationship is to be considered as truly interactive. The pain reaction will depend on the individual's appraisal of the pain experience and his or her ability to cope with nociceptive stimulation (link g). Pain tolerance and threshold may be directly affected by those factors (cf., Turk et al., 1983). However, the stress of continuous pain will most likely diminish the individual's ability to cope with new stressors (link m) thus feeding still more into the stress-tension-pain cycle.

In the long run, sustained muscular contractions may result in irreversible degeneration and atrophy of the muscle fibers (cf., Fassbender, 1973; 1975). Display of pain may motivate reinforcement of pain behaviors (e.g., expression of pain, immobility - links n, o -), and operant conditioning of pain behaviors may develop as an additional complicating factor.

The importance of the model lies in the specific theoretical predictions and its implications for assessment and treatment of CBP. General predictions of the model are listed below. Concrete experimental hypotheses resulting from the model will be presented at the end of this chapter (see 4.4.).

The general predictions are that:

(a) CBP patients have higher resting tension levels (and vasoconstrictions), as well as higher and longer muscular reactions (and vasoconstriction) to relevant stress stimuli, than healthy controls;

(b) the increased muscle tension (and vasoconstriction) is specifically related to the back muscles;

(c) this physiological hyperreaction stems from an insufficient ability to cope with emotional stress, given vulnerability of the back;

(d) the increased muscle tension (and ischemia) is a final common pathway for CBP;

(e) CBP patients have higher anxiety levels than healthy controls, especially when anticipating movement;

(f) CBP patients show poor stress coping abilities;

(g) CBP patients have decreased spinal mobility compared to healthy controls;

(h) operant processes moderate these relationships.

4.3. Empirical Evidence for the Model

Before presenting an experiment that tests various key aspects of the model, the available evidence for the assumed stress-tension-pain-anxiety-immobility-tension-pain relationship will be summarized. The evidence cited in the section on muscular processes in CBP, and the data on the psychoanalytic, respondent, modeling and cognitive-behavioral models, are also relevant here and will be discussed in more detail.

4.3.1. Research on the Muscle Tension Levels of Chronic Back Pain Patients

Only a few studies have compared the muscle tension levels of chronic back pain patients and healthy controls. Grabel (1973) constrasted the EMG-levels of the back muscles (erectores spinae group) of 3o low back pain patients with those of 3o healthy controls. The resting levels as well as the muscular reactions to an experimentally induced stressor (threat of shock) were tested. He found a highly significant group difference in the resting level condition between the groups,

with the values of the low back pain patients much higher than those of the control subjects. The muscular reactions to stress were not different between the groups. It is, however, questionable whether the stressor was relevant for the subjects. Back patients might overreact only to certain idiosyncratic stressors, and stimulus-specificity (cf., Wenger & Cullen, 1958) might also play a role. Perhaps return to baseline tension levels would be the more relevant variable (cf., Philips , 1977); this was, however, not measured. Grabel also failed to take recordings from other muscle sites; therefore, no conclusion about the specifity or generality of the elevated tension levels can be made. A more critical problem is, that he used a highly questionable method to measure EMG. The number of peaks exceeding a value of 1o microvolts was used as the EMG-measure, which is highly unusual for psychophysiological measurements, and which completely disregards the more frequently occurring lower levels. In addition, Grabel (1973) selected only those patients without organic findings and without palpable muscle spasms.

Kravitz et al. (1981) compared the EMG values of the back muscles of 22 low back pain patients to those of healthy controls during a resting baseline and during differential relaxation. Differential relaxation is described as the tensing of certain muscles while keeping other muscles relaxed. Low back pain patients tensed their back muscles more than healthy controls when instructed to tense those muscles minimally. The tension levels of the patients' back muscles were slightly but not significantly higher when they tensed other muscle groups, although Kravitz et al. assert that the increased tension is indeed significant (p. 172). They did not find higher resting tension levels, in contrast to Grabel. They also failed as he did, to take measures from other muscles to examine whether back pain patients exhibit generalized or back-specific muscular tension.

Hoyt et al. (1981) compared the paravertebral EMGs (rectus abdominus and erector spinae muscles) of low back pain patients and healthy controls during 1o-minute periods of sitting, standing, and in a semi-Fowler position. They found increased EMG levels in the erector spinae muscles of the low back pain patients only while standing.

Collins et al. (1982) compared 11 low back pain patients and 11 healthy controls in their frontalis EMG, heart rate, and skin conductance levels, in a variety of conditions: a 1o-minute sitting baseline period, 36 seconds (3 x 12-second trials) each of standing, 45-degree bend, maximum bend, forward pelvic tilt, backward pelvic tilt, unsupported sitting,and supine knees bent, and then during 5 minutes of mental arithmetic (sitting) and 3 x 12-second trials of cold pressor test (sitting). They found no differences in the resting tension levels of the erector spinae muscles between the low back pain and the healthy subjects. The patients' frontalis levels were, however, elevated, as were their overall skin conductance levels. Contrary to expectations, they found reduced paraspinal EMG levels in the patients during forward pelvic tilt and the 45-degree bend. This finding partially confirms Basmajian's (1974, 1978) statements that low back pain patients show, in fact, lower muscle tension, though he never provided clear evidence for this assertion. This finding might be explained by the fact that in some painful conditions palpable muscle spasms do not produce any EMG activity (Holt, 1966; Kraft et al., 1968). The absence of EMG activity may also stem from the method of measurement. Cobb et al. (1975) demonstrated that needle electrodes measure too selectively and may not be suitable for recordings from areas with widely-ranging tension levels. They also pointed out the importance of a high amplifier sensitivity (1o microvolts/cm) and integration of the EMG signal.

The Collins et al. study (1982) does not describe the amplification of the signal. It also fails to indicate CBP sample characteristics, thus not allowing any conclusions about the organic or psychological make-up of these patients. The researchers also fail to report the standard deviations of their patients' EMGs. From our own pilot work we know that CBP patients show widely-divergent EMG reactions with very heterogeneous and instable variances, which may hide any true effects. Collins et al. (1982) also fail to take into account the initial values of their sample which are important determinants of the stress response (cf., Wilder, 195o). Keefe et al.(Note 2) report that about 3o % of their back pain patients show generalized muscle overactivity, in, for example, the frontalis, trapezius, forearm extensor, and hip adductor muscles in various positions. They failed, however, to record at the back itself because many patients had had surgery (Keefe, Note 3), which

may cause highly variable denervation potentials (cf., Lippold, 1967).

A different approach toward assessing the relationship of CBP and muscle tension was used by Wolf and his collegues (cf., Jones and Wolf, 1980; Wolf and Basmajian, 1977; Wolf et al., 1982). They believe that assessment of muscular activity during dynamic movement may be more important than measurement during resting states. They assert that CBP patients show imbalance and assymmetry of paravertebral muscle activity without, however, providing more than casual examples. Cram and Steger (Cram, Note 4; Cram & Steger, Note 5) suggested that unilateral recordings (separate for both sides of the back) are essential for the lumbar muscles. They emphasize that assymmetries of the sides of the back may be important in the diagnosis of back syndromes.

The studies discussed in this section provide suggestive evidence for the role of muscular deficiencies in CBP. Unfortuanetly inadequate designs and methodological weaknesses (for example, even the most recent study (Collins et al., 1982) shows major flaws, as discussed above) preclude classification of these studies as any more than pilot research. It is therefore necessary to examine, via a more adequate methodology, the psychophysiological characteristics of back pain patients in comparison to various other groups. These groups should include healthy and other patient controls (this allows an answer to the question whether any observed abnormalities are typical for CBP patients, or are characteristics of any pain patient). The CBP group should also be carefully described and classified into subgroups with "clear degenerative changes", "unknown pathology", "musculo-skeletal" and so forth, to determine the role of organic factors in the pain problem. The evaluation needs to be much more comprehensive and needs to include multiple psychophysiological measures. These measures need to be taken under various stress and non-stress conditions, using adequate recording techniques, i.e. unilateral recordings with surface electrodes, high sensitivity, and so forth.

4.3.2. Research on the Stress-Emotion-Tension Relationship

Although a number of authors assume that emotional events do influence muscle tension and can eventually cause pain, the relationship between emotional stress and muscular hypertension is still not sufficiently clarified.

Most of the research that has been done has heavily depended on psychoanalytic theory and has been conducted in the 195o's and 196o's, based mostly on single cases, lacking control groups and statistical sophistication (see section 3).

Most of these studies dealt with muscle tension headache and the relationship of emotional stress and muscle tension in these patients (e.g., Malmo & Shagass, 1949; Shagass & Malmo, 1954; Sainsbury & Gibson, 1954). The results of this research are equivocal (e.g., Gannon et al., 1981). Dorpat and Holmes (1962) (see also Holmes & Wolff, 1952) compared the EMG values from the lumbar sacrospinalis, trapezius and hamstring muscles of 65 back pain patients and 1o healthy controls, and found elevated values of these muscles in the patients when performing simple tasks and discussing personal conflicts in a psychoanalytic interview. They assume that the CBP patients show a generalized muscular hyperreaction to stressful stimuli. Janus (1975) interviewed patients with neck and shoulder pain and found dramatic EMG increases in the trapezius muscle when aggressive impulses were discussed, or when the patients were confronted, and decreases, when fantasies (especially flight fantasies) were verbalized. The lack of sufficient control conditions makes these results questionable, however. As discussed previously, Collins et al. (1982) subjected 11 low back pain patients and 11 healthy controls to two different stress conditions: the cold pressor test, and mental arithmetic. Under both conditions the low back pain patients showed higher frontalis muscles tension than the controls, but their paravertebral muscle tension levels were not significantly different. Collins et al. (1982) interpret these findings as a sign of generally higher arousal in the low back pain patients resulting from chronic low back pain.

These results need replication. Also, a variety of stressors should be used, and their relevance for the subjects should be tested. It is also important to measure from many different muscle sites. The Dorpat & Holmes experiment does not meet that requirement, since all of their measurements were close to the back. A series of studies (see Bradley et al., 1981; Crown 198o for a review) has evaluated the MMPI profiles of CBP patients in order to detect emotional disturbances in these patients, and to use them in prognosis. The research revealed that there are subgroups of CBP patients with definite psychopathology (e.g., Brad-

ley et al., 1978; Sternbach et al., 1973b). No studies relating those emotional disturbances to muscle tension levels or reactivity have yet been conducted.

4.3.3. The Relationship between Tension, Immobility and Pain

The findings in this area are quite limited. In some of the above cited studies an increase in muscle tension following emotional stress was noted, but without a concomitant onset or increase of pain. The time lag for pain to develop, however, might be longer than the assessment time in these experiments.

Muscular hypertension has been shown to invoke pain. Wolff (1963) induced pain in the head experimentally by applying continuous pressure around the head, or injecting histamine. He could show that a strong muscular contraction led to pain that was relieved by massage and increased by movement. He used, however, only small numbers of subjects and provided no statistical data. Indirect evidence for a relationship between pain and tension can be attributed to the reports of Malmo et al. (1955) and Sainsbury et al. (1954), who found an increase in EMG values when headaches increased. These studies, however, focus only on tension headaches, and it has lately been heavily disputed whether there is any relationship at all between muscular tension and "tension" headaches (see Philips, 1977; Gannon et al., 1981). It is an open question to what extent headaches and backaches do have comparable underlying psychophysiological mechanisms. Practically no research about the tension-pain relationship has been done in back pain. DeVries (1976) compared the EMG and pain levels of eight low back pain patients and four healthy controls during a 22-minute standing phase. Three of the low back pain patients developed back pain while standing, which was accompanied by considerable EMG increases. Pope et al. (1981) examined the relationship between spinal mobility and pain tolerance in 89 low back pain patients, but unfortunately did not measure their EMGs. They found 2 subgroups of patients: 1) a group with high pain tolerance and high spinal mobility and 2) a group with both low pain tolerance and low spinal mobility. The latter seems to predict unfavorable outcome for the long-term functional capacity of the individual. If one sees spinal mobility as related to higher muscle tension, these data might give indirect evidence to a tension-pain relationship. In an ana-

logue study, Cobb et al. (1975) injected sodium chloride into the left wrist extensor groups and the lumbar area (L1) of seven subjects. This procedure caused sharp increase in pain and EMG levels in all but one of the subjects. They consider their findings as supportive of a pain-spasm-pain concept of CBP.

Another line of evidence comes from studies on the efficacy of EMG biofeedback for chronic low back pain. Although the results are equivocal , a number of studies demonstrated the efficacy of EMG reduction in the paraspinal muscles for attentuation low back pain (e.g., Flor et al., 1983; Nouwen & Solinger, 1979). However, additional cognitive factors seem to be involved, since Nouwen & Solinger (1979) could show that at 6 months' follow-up their patients retained their pain reduction, but had acquired the old - higher - EMG levels again.

In summary, it is not yet sufficiently clarified to what extent muscular hypertension and pain are related, nor is it clear whether resting muscle tension or reactivity to stress, or delay in the return to baseline, are the key variables; nor are the time-lags of these processes sufficiently known.

4.4. Purpose of the Study and Key Hypotheses

This study was designed (see section 5 for a full description) to investigate the relationship between stress and psychophysiological activation in chronic back pain. It is hypothesized that CBP has multiple influences that interact within a diathesis-stress model. The goal of this study is to identify and more fully investigate these sources of influence and to determine their course of interaction, using an improved methodology in manipulating psychological variables as well as measuring physiological variables.

Chronic back pain patients, pain patients suffering from other pain syndromes, and healthy controls are be exposed to personally relevant as well as general stressors and neutral stimuli.The tests consist of a 10-minute resting baseline and 4 stress tasks, each consisting of a 1-minute pretrial baseline, a 1-minute trial, and a 5-minute inter-trial interval. The stress-situations consist of (a) talking about a recent stressful episode (= personally relevant stressor), (b) talking about a recent pain episode (= personally relevant stressor), (c) reciting the alphabet (neutral talking condition), and (d) working through given men-

tal arithmetic (general stressor).

The study specifically addresses the effects of: (1) baseline muscular levels in contrast to EMG elevations in response to stress, and delayed return to baseline following stress; (2) personal relevance versus generality of stress; (3) general psychophysiological reactivity to psychological stress, in contrast to specific activity at the site of pain.

The following 10 experimental hypotheses are to be investigated, with hypotheses 1, 2, 3, 8, and 9 as the central hypotheses of the model.

1. CBP patients are expected to show greater increase in paraspinal muscle activity when exposed to personally relevant stressors than are other pain patients or healthy controls due to a response stereotypy of their back muscles (cf., Lacey & Lacey, 1958). They are expected to show only minimal back muscle responses not different from those of the general pain and healthy control group when exposed to a general stressor (arithmetic) or a neutral condition (alphabet). The EMG response is expected to be especially marked at the affected side of the back, if the pain is unilateral.

2. CBP patients are expected to exhibit a long duration period in the return to the baseline of their paravertebral muscle tension levels after stress, as compared to other pain patients and healthy controls. It may be hypothesized that a long delay before returning to baseline is instrumental in the development of chronic muscular hypertension and pain (cf., Phillips, 1977). The delay is expected to be longer at the affected side of the back if the pain is unilateral.

3. The resting tension levels in the back muscles of the CBP patients may be elevated compared to those of other pain patients and healthy controls. This assumption must be specified. Basmajian (1976) has emphasized that spastic muscles may not always exhibit elevated EMG levels. The research of Passmander (1973, 1975) would suggest that the longer the pain problem has existed, the more atrophy of the muscles may have occured, and the lower the EMG potentials that may be measured. This hypothesis is therefore not as central to the study as hypotheses 1 and 2. In addition, the paraspinal resting tension levels are expected to be higher at the affected side of the back if the pain is unilateral, and they are expected to be comparable for

both sides if the pain is bilateral.

4. No difference between the muscle tension levels as well as muscular reactions to stress of CBP patients with or without known pathology is expected. Although injury to the back may lead to the initial acute pain, it may eventually become a predisposing physiological condition and/or a personally relevant stressor for the CBP group. No paravertebral EMG difference between patients with or without surgery is expected. It is rather hypothesized that differences in paraspinal EMG reactivity or levels are related to psychological variables.

5. It is hypothesized that the CBP patients can be distinguished from the general pain patients and the healthy controls on grounds of their back muscle reaction to the personally relevant stressors. Psychological treatment focussing on this psychophysiological hyperreaction to stress is believed to lead to an attenuation of the muscular stress response.

6. All groups are expected to show a marked frontalis reaction (amplitude, delay in return to baseline) when exposed to a general stressor (mental arithemtic), with the pain groups, who are more stressed due to their pain state, exhibiting an even higher reaction. The frontalis reaction is expected as a stimulus-specific response and as a general stress response. Therefore, all groups are also expected to show (though less significant) frontalis tension increase during the pain and personal stress situation, with the pain groups showing a stronger reaction than the healthy controls.

7. All groups are also expected to show high heart rate and skin resistance level responses when exposed to the personally relevant or general stress situations, with the pain groups showing a greater reaction than the healthy controls. However , when discussion of a recent pain experience is used as a relevant stressor, the healthy controls are not expected to display a marked reaction,since this is not indicative of relevant stress for this group. Although mental arithmetic is not considered to be as potent a stressor as the personally relevant stress situations, the additional mental effort required to perform the task was hypothesized to increase the arousal level of the subjects to levels similar to that of the pain and stress con-

ditions.

8. CBP patients are expected to show spinal immobility, which is positively correlated with muscle tension, state anxiety and pain levels.

9. The subjective pain, tension, unpleasantness and effort ratings after the experimental conditions are expected to increase in the stress conditions (especially the personally relevant condition) thus differentiating them from the neutral control condition. The two pain groups are expected to display higher pain, tension, unpleasantness and effort ratings than the healthy controls. The effort ratings are expected to be highest in the mental arithmetic condition in all groups. The state anxiety scores are expected to be elevated in both pain groups as compared to the healthy controls.

1o. CBP patients with high stress responses (high amplitude, long delay in return to pretrial baseline levels of the paravertebral EMG) are expected to show high scores of helplessness (as measured with the Pain Experience Questionnaire) and depression, low scores for coping with pain (as assessed by interview and high scores for the evaluative and affective components of the McGill Pain Questionnnaire), high general psychopathology ratings (interview), and high external and low internal health locus of control scores , as indicators of coping ability and appraisal of stressful (painful) stimulation.

5. Methods

5.1. Subjects

The CBP group (CBP) consisted of 17 patients who had had a CBP complaint for more than 6 months and had come for treatment to the Pain Management Program (PMP) at the West Haven Veterans Administration Medical Center (VAMC). Patients suffering from back pain of inflammatory origin or patients with neurological complications (according to the criteria of Blumberg et al., 1964) were excluded. All patients who had been suffering from primary back pain for more than 6 months with diagnoses ranging from "CBP of unknown origin" to "spondylolisthesis" were accepted for the study. The general pain group (GP) consisted of 17 patients suffering from various pain syndromes unrelated to the back for more than 6 months. They were also patients of the PMP. The healthy control group (H) consisted of 17 volunteers who were recruited from the hospital staff, were outpatients of other clinics,or friends of the investigator. The patients of the PMP participated in the study as part of their assessment procedure. The other subjects were asked to volunteer when they came to outpatient clinics or after responding to notices pinned on the walls at the VAMC (see Appendix A-1 and A-2). The groups were matched for age, sex, and socioeconomic status (defined by profession and education). Non-CBP patients with a history of CBP though not currently receiving treatment or with acute back pain at the time of the study were excluded, as were individuals currently receiving psychiatric treatment.

65 patients or control subjects had originally volunteered. Of those, 4 subjects (1 CBP, 1 GP, 2 H) had to be excluded due to malfunction of the equipment, 3 GP patients and 4 controls were excluded because they suffered from acute back pain or had a history of CBP, 1 CBP patient was excluded because of extremely variable baseline EMG (probably due to denervation) and 2 patients (1 CBP, 1 H) were excluded due to lack of compliance with the instructions. Table 1 summarizes the demographic data and Table 2 gives information on the disease characteristics of the subjects. The subjects in the three experimental groups were comparable in regard to sex, age, marital status, education and employment status (no significant differences, see table 1), although the healthy group contained more employed and fewer unemployed or retired subjects than

the two pain groups. The two pain groups were very similar in their demographic variables, as well as in the duration of the pain problem, disability and number of pain-related surgeries (see tables 1 and 2).

TABLE 1

SUBJECT DEMOGRAPHIC CHARACTERISTICS BY GROUP

	Chronic Back Pain	General Pain	Healthy Controls	Significance test
Sex male	12	15	13	X^2_2=1.623
female	5	2	4	p^2= .444
Age in years				
mean	47.oo	49.93	45.18	$F_{2,47}$=.546
s.d.	(15.52)	(13.94)	(14.42)	
range	27-73	29-68	23-64	p = .583
Marital Status				
single	2	2	3	X^2_4= 2.228
married	13	12	12	p = .694
widowed/ divorced	2	3	2	
Education in years				
mean	13.11	12.35	14.23	$F_{2,48}$= 1.272
s.d.	3.1o	3.33	3.9ò	
range	8-21	7-21	5-22	p = .289
Employment Status				
retired	6	6	2	X^2_8=6.67o
disabled	3	3	4	
unemployed	2	2	o	p = .573
employed	5	5	9	
in training/ student	1	1	2	

TABLE 2

SUBJECT DISEASE CHARACTERISTICS BY GROUP

	Chronic Back Pain	General Pain	Significance test
Duration of Pain in years			
mean	1o.79	8.93	t_{28}=.72
s.d.	13.o9	8.37	
range	8 months to 38 years	1 year to 3o years	p=.477
Etiology			
Intractable Pain and Cancer	-	1	
Rheumatic Disease	-	7	
Headache	-	3	
Neck and Cervicobrachial Neuralgia	3	-	
Low Back Pain and Sciatica	14	1 (only hip affected)	
Peripheral vascular disease	-	1	
Nerve Compression Syndrome/	-		
Painful Sears/Postherpetic Neuralgia	-	1	
Stump pain	-	1	
Visceral pain of the chest/abdomen	-	2	
Site of pain			
upper back	3	-	
lower back	14	-	
head	-	3	
Stomach, pelvic	-	1	
arm, hand	-	3	
legs, feet	-	6	
multiple joint	-	1	
chest	-	2	
hip	-	1	
Surgery (pain-related)			
yes	8	6	$X^2_{(1)}$=.486
no	9	11	p = n.s.
Kind of Surgery			
laminectomy	5	-	
fusion	1	1	
both	2	-	
other	o	5	

TABLE 2 cont.

	Chronic Back Pain	General Pain	Significance test
Number of Surgeries			
o	9	11	$X^2_{(2)}=1.334$
1	3	4	
≥ 2	5	2	p = n.s.
X-Ray diagnosis			
no damage	7	-	
slight	1	-	
moderate	6	-	
severe	3	-	
Diagnoses in the CBP group (medical)			
herniated or ruptured disc	3	-	
degenerative disc/joint disease/spondylosis	7	-	
Low Back Pain- Sciatica	1	-	
LBP of unkown or muscular origin	4	-	
chronic unstable CBP syndrome	1	-	
Spondylolisthesis	1	-	
Compensation (pain-related)			
yes	9	8	X^2=.255
no	8	9	p=n.s.

The general pain group suffered from a variety of pain complaints, for the most part arthritis, with the feet, legs, arms and hands as predominant pain sites. The CBP group consisted of 3 upper back and 14 lower back pain patients, with diagnoses of spondylosis or degenerative disc disease (N = 7), herniated or ruptured disc (3), low back pain of unknown or muscular origin (4), sciatica (1), chronic unstable low back pain syndrome (1) and spondolylisthesis (1). 8 patients in this group had undergone back surgery with 5 of them experiencing multiple operations. 9 patients showed moderate to severe, 8 no to slight degenarative changes as judged by their x-rays (physicians' diagnoses). The patients in the CBP group had suffered from CBP for an average of 1o.79 years (sd = 13.o9), with pain duration ranging from 8 months to 38 years. The majority of the patients in this group were severly affected by their pain problem. Only 6 out of 17 were employed or in training (students), 11 were disabled, unemployed or retired due to their pain problem and 9 (53 %) received pain-related compensation. This CBP sample is thus

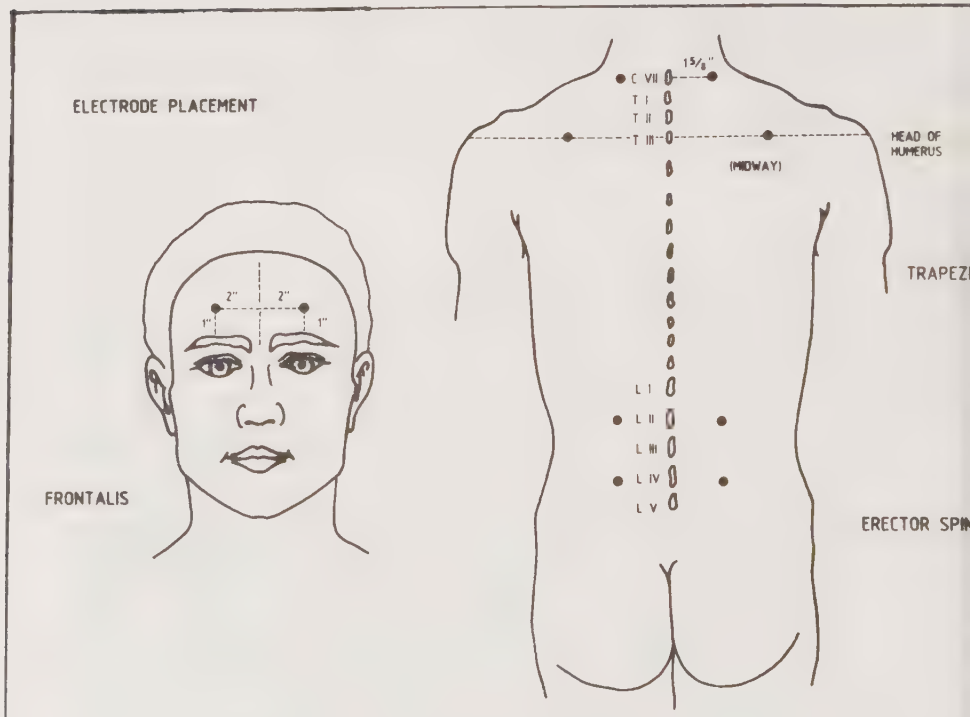
comparable to the severly affected CBP patient seen in other settings (cf., Gottlieb et al., 1977; Keefe et al., 1981).

5.2. Instrumentation

All physiological measures were recorded by a Cyborg biolab 11. The biolab is an 8-channel physiograph, operated by an Apple II Computer. Only 5 channels were available for the recordings of this study.

EMG measures were taken from the frontalis and the erector spinae (low back pain) or trapezius (upper back pain) muscles using standard electrode placement (cf., Lippold, 1967). Figure 2 shows the electrode placements used in this study.

FIGURE 2



For the frontalis bilateral placement was chosen because the frontalis does not seem to display marked side asymmetries (see Cram, Note 4). The trapezius and erectores spinae measurements were taken separately from both sides of the back because side asymmetries might be significant for the back musculature (cf., Cram, Note 4). Fifteen mm AgAgCl sensors filled with Spectra 360 (Parker La., Inc.) electrolyte were attached with adhesive discs and the signal was fed into a remote amplifier. The skin was cleansed with 70% alcohol, and a mild abrasive (Redux, Hewlett-Packard) was applied to the skin. Root mean sqare (RMS) signal processing was used with an input noise level below .2 microvolts RMS. Further parameters of the system were: common mode rejection above 90 decibels, input impedance above 50 megaohms, input current below 0.7 nanoamps,bandpass filter 100 to 250 Hz, sensitivity range 0 to 100 microvolts. The signal was processed at a sampling rate of 10/sec and the digitized signal was averaged for 2-second intervals and stored on floppy discs.

Heart rate was measured by a photopletysmographic sensor attached to the second finger of the subject's dominant hand. The modulated sensor light was transformed into an electric signal that was amplified, counted and averaged. Beats per minute were stored on disc for further processing.

GSR was measured via 4 mm AgAgCl sensors plugged into a junction box feeding into the physiology module. The sensore were filled with surgical jelly (Surgilube, Fongera & Co.) and attached to the subject's nondominant hand (at the thenar and hypothenar eminences of the palm, cf., Hassett, 1977), after it had been cleansed with soap and water. D.C. current was used with the magnitude of the current in the range of 5 microamps/cm^2 . The module was calibrated via a resistance substitution box. Analog to digital conversion was applied to the data with a high resolution ($\pm$ .1 Volt to $\pm$ 5 Volt conversion,values from 0 to 4095) and high amplification gain (50).

5.3. Experimental Conditions

A three by four design (3 groups by 4 experimental conditions) was employed to test the hypotheses delineated in section 4.4 (see table 3 for a summary of the experimental design).

TABLE 3

EXPERIMENTAL DESIGN

		Conditions											
		Control			General Stress			Personally Relevant Stress					
Groups	Resting Baseline	Neutral Talk (Alphabet)[1]			Mental Arithmethic[1]			Stress Talk[1]			Pain Talk[1]		
		BL	TR	P[2]	BL	TR	P[2]	BL	TR	P[2]	BL	TR	P[2]
Chronic Back Pain (N=17)	— — —	— — —			— — —			— — —			— — —		→
General Pain (N=17)	— — —	— — —			— — —			— — —			— — —		→
Healthy Controls (N=17)	— — —	— — —			— — —			— — —			— — —		→

BL = Pretrial Baseline TR = Training P = Posttrial Baseline

[1]The conditions were counterbalanced

[2]Each condition consisted of a 1-minute pretrial baseline, 1 minute exposure, and 5 minutes posttrial baseline.

A 10-minute resting baseline was used to determine the subjects' baseline muscle tension, heart rate and skin resistance levels. Then the four experimental conditions were presented in a predetermined and counterbalanced order of appearance. Each condition lasted for 1 minute and was preceded by a 1-minute pretrial baseline, and followed by a 5-minute posttrial baseline.

For the resting baseline, the subjects were instructed to sit still, rest, and avoid movement for a 10-minute period. No other instructions were given.

For the neutral talking condition, subjects were asked to recite the alphabet for one minute at any speed they wished, and they were asked not to move during the task. If a subject was unable to perform this re-

requirement, repeated counting from 1 to 3 was substituted.

For the first personally relevant stress situation subjects were asked to recall a stressful event from the last week, or from further back if necessary. They were asked to select an experience with a stress rating of at least 5 on a scale from o (not at all stressful) to 1o (very stressful). They were to remember it as vividly as possible and to talk about the experience for one minute.

For the second personally relevant stress situation subjects were asked to recall a recent pain episode (from the last week, or further back , if necessary) , with a pain intensity of at least 5 on a scale ranging from o (no pain at all)to 1o (extreme, very severe pain). They were asked to recall the pain experience in as much detail as possible and to talk about it for one minute.

For the general stress situation subjects were asked to count backwards in steps of seven (serial sevens) starting at 758; they were to count as quickly as possible. Subjects who were unable to perform this task were asked to count backwards by steps of 3 or , if still unable, to count backwards by 3 or 1 from 1oo. Subjects were not prompted if they lost track of their position.

The experimental conditions were selected according to the following reasoning: the recitation of the alphabet served as a control for the effects of mere talking on the various physiological variables.Since it was hypothesized that only those situations that were acutally experienced as very stressful by the subjects had an emotional impact on them, the choice of the personal stress conditions was left to the subjects. They were asked to choose a situation they themselves defined as stressful and had experienced only recently. They were also asked to choose a recent pain experience, under the assumption that pain acts as a potent stressor in pain patients. Talking about a stressful event was chosen as the preferred mode of stress-induction. Direct exposure to a relevant stressor would have required extensive pre-experimental assessment, as well as a large organizational effort to produce the stress. Talking about a stress event was preferred to imagery because it is closer to everyday interaction than is elicitation of an image. Mental arithmetic was chosen as a second control condition. It was considered a general stressor because it had not been chosen by the subjects as

a stress situation relevant to them. However, mental arithmetic of this type has been shown to produce stress and is frequently used in laboratory studies of stress (cf., Collins et al., 1982). It is hypothesized that mental arithmetic is stressful because it requires mental effort, but that it does not elicit the emotional involvement which interpersonal or pain-related stress may produce.

A stress exposure of one minute was found during pilot work to be sufficient. The subjects (mainly army veterans) were not very verbal, and longer talking times did not prove to elicit more stress. The 1-minute pretrial baseline was introduced because the CBP patients (but also some of the other patients) were unable to sit still without moving during the entire 45-minute recording period. The subjects were therefore given the opportunity to change their position between the trials. This required new baselines for each trial. The 5-minute posttrial baseline was necessary to allow the physiological values to return to pre-trial baseline levels, thus preventing carry-over effects. Instructions for the tasks were given after the 1-minute pretrial baseline. Pilot work had shown that patients would start to work on the tasks already during the pretrial baseline if instructions were given before, and the subsequent reaction in the trial would be attenuated. There was no break - only a beep signal - between the end of the trial and the posttrial baseline. This allowed accurate measurement of the delay in the return to baseline of the various measures.

For the entire experimental procedure and resting baseline, sitting in a recliner with straight back was the required body position. This position was chosen because it is very close to positions the patients habitually assume during the day, and it requires some back muscle activity (cf., Floyd & Silver, 1955). Lying in a prone position was rejected as being a very relaxed and unusual daily position. A supine position could not be used because the sensors had to be attached to the back and standing was too strenuous for the long procedure (45 minutes). It was not possible to take the recordings in various body positions due to the long duration of the experimental conditions.

Before and after each condition pain, tension,stress, and effort ratings were taken. The State form of the State-Trait-Anxiety Inventory (Spielberger et al.,197o) was given before the baseline and after the last experimental condition. These measures will be described in detail

in the next section.

5.4. Dependent Variables

Dependent measures were collected on the biological, motor, and verbal-subjective levels (cf. Birbaumer, 1977; see table 4 for an overview).

5.4.1. Physiological and Radiological Measures

The subjects' paravertebral and frontalis EMGs, their heartrate and their skin resistance levels were assessed during the 10-minute resting baseline, as well as during the pretrial baselines, the trials, and the posttrial baselines. The baseline levels were used for the group comparisons of tonic EMG, heartrate, and skin resistance levels. The reactivity to the stress conditions was measured as the difference between pretrial and trial levels. Latency in return to pretrial baseline levels was measured in seconds. It was hypothesized that the change from pretrial to trial levels and also the delay in the return to baseline levels would be important indicators of abnormal reactivity of a physiological measure (cf., Gannon et al,, 1981).

The paravertebral EMGs were taken to determine the existence of a response stereotypy in these muscles. Surface electrodes and unilateral recordings were used following the suggestions of Cobb et al. (1975) and Wolf et al. (1982). Frontalis EMG was recorded to determine the specificity of the response for the back muscles and also because frontalis activity may be a general indicator of stress (cf., Passchier & Helm-Hylkema, 1981). Additional measurement of forearm muscle tension would have provided still better determination of the specificity of a back muscle reaction, since forearm tension would not be expected to increase during exposure to emotional stress. However, equipment limitations did not allow this measurement. The frontalis EMG was also used to examine the hypothesis of stimulus-response specificity of the mental arithmetic task.

Heart rate and skin resistance levels were measured as general indicators of stress and CNS activation (cf., Lang, 1971).

To determine the extent of degenerative changes in the CBP patients, x-rays of the lumbar or cervical spine were evaluated. The changes were

TABLE 4
ASSESSMENT INSTRUMENTS USED IN THE STUDY

Variable	PMP Assessment (about 1 week before the experiment)	Beginning of Session	Pre Baseline	Post	4 Experimental Conditions (same measures in each) Pre Baseline	Trial	Post-Baseline	Post	End of Session
EMG left back			x		x	x	x		
EMG right back			x		x	x	x		
EMG frontalis			x		x	x	x		
Heartrate			x		x	x	x		
Skin resistance level			x		x	x	x		
Finger to Floor Distance		x							
Pain Behavior Checklist	x								
Pain-Rating			x	x				x	
Tension-Rating			x	x				x	
Effort-Rating			x	x				x	
Stress-Rating			x	x				x	
State Anxiety	x	x							x
McGill Pain Questionnaire	x^1 or x^2	x^1 (first week)							
Pain Rating Cards	x^2								
Depression Adjective Check List	x^2								
Beck Depression Inventory	x^2								
Health Locus of Control	x^1 or	x^1							
Pain Experience Questionnaire	x^1 or	x^1							
Locke-Wallace Marital Adjustment Scale	x^2								
Pain Interview	x^2								
Medical Data	x^1 or	x^1							

[1] = measures were only given to ss in the pain groups [2] = measures were only given to PMP participants

rated as slight, moderate, or severe according to the physician's reports in the medical records, on the radiological evaluation of these patients. The x-ray reports were rated by the investigator before the patients' physiological reactions were evaluated. The kind and number of surgeries as well as medical diagnoses were also taken from the medical records, as well as from an interview.

5.4.2. Behavioral Measures

Spinal mobility was assessed by a tape measure. Subjects were asked to reach with their fingertips as far as they could to the floor while keeping their knees extended. The finger-to-floor distance was measured in centimeters. This score is a common and convenient measure of mobility of the lower back area (cf., Albrecht, 1975; Moll and Wright, 1980).

In addition, the PMP therapists scored the pain behaviors of the patients who participated in treatment on a pain behavior checklist (PBC, Turk,Note 6,see Appendix B-1). The PBC is a 23-item list of commonly displayed pain behaviors (cf., facial grimacing, moaning, irritability). The therapist rates the frequency of occurrence of the behaviors (1 = definitely does not exhibit behavior; 2 = exhibits behavior occasionally; 3 = exhibits behavior regulary). It is used as a general index of the behavioral component of pain in the patients. The reliability and validity of the scale are currently being investigated.

5.4.3. Measures of the Verbal-Subjective Level

A number of rating scales, questionnaires and a pain assessment interview were used to evaluate various aspects of the patients' pain experience, their reaction to stress, and their general emotional, social and vocational adjustment. In addition to the measures used for the experiment all pain patients who also participated in the Pain Management Program at the VAMC (28 of the 34 members of the two pain groups) filled out the assessment instruments of the PMP, which were also used in this study. An overview of the instruments used in this study can be found in table 5.

5.4.3.1. Pain Measures

The PMP patients completed daily pain-monitoring cards (cf., Budzynski et al., 1973; Philips, 1977) for one to two weeks prior to the experimental session. They were asked to rate their pain hourly, on a scale from 0 (none, no pain) to 5 (excruciating, incapacitating, intense, severe pain - anchored according to Melzack, 1975). These pain rating cards are widely used in clinical pain studies, also with CBP patients (cf., Nouwen & Solinger, 1979). The hourly ratings are added up and divided by the hours that are recorded per day (cf., Philips, 1977). Thus a daily pain average is computed. The average of a week of pain monitoring prior to the experiment was used as a global indicator of average pain intensity (see Appendix B-2).

In addition, the pain patients completed the McGill Pain Questionnaire (MPQ, Melzack, 1975). The MPQ contains 78 ranked adjectives providing quantitative data on the sensory, affective and evaluative qualities of the pain experience, as well as a pain-intensity rating scale and descriptions of temporal qualities of the pain. Dependent measures used in this study were the Present Pain Intensity (varying from 0 = no pain to 5 = excruciating, horrible pain, see Appendix B-3), the word group chosen to denote the temporal quality of the pain, and the average pain rating index for each of the components of the pain experience (sensory, affective, evaluative, other) and for the total score. Reliability and validity of the MPQ have been sufficiently established (cf., Byrne et al., 1982; Melzack , 1975; Reading, 1979). It has been shown to be sensitive to the intensity and quality of both clinical (cf., Melzack & Perry, 1975) and laboratory pain (cf., Klepac et al., 1981).

During the experiment all subjects were asked to give present pain ratings on a scale ranging from 0 (no pain) to 10 (horrible, excruciating pain) (see Appendix B-4). The ratings were taken before and after the initial baseline, and after each of the experimental conditions.

5.4.3.2. The Pain Assessment Interview

The Pain Assessment Interview (Report) contains detailed questions on

the nature of the pain problem, variables affecting it, the patient's past and present use of medication, alcohol and/or illicit drugs, the treatments the patient has already undergone for his or her pain problem and their effectiveness.The vocational, marital, sexual and social situation of the patient is assessed, as well as the impact the pain has had on these areas. In addition, neurovegetative problems are assessed. The patient's current mental status, his or her coping skills, and current or previous psychological tratment and its effectiveness are measured. Most answers are rated on a 4-point scale (0 = not applicable; 1 = little, 2 = moderate, 3 = very much; see Appendix B-5). The interview gives an impression of the impact the pain has had on various areas of the patient's life, and the role of job, family or social relations with regard to the pain problem.

5.4.3.3. Additional Verbal-Subjective Measures

The present anxiety level of the subjects was assessed via the State form of the State-Trait-Anxiety Inventory developed by Spielberger (Spielberger et al., 197o). It contains 2o statements about a person's present level of anxiety. They are rated on a 4 point scale (1 = not at all to 4 = very much so). It is a reliable and valid instrument to assess state anxiety levels (cf., Kendall et al.,1976). It was administered to the pain patients at the first PMP assessment prior to the experiment, and then to all subjects before (following instruction about the tasks) and after the experimental session. The inventory was used as a measure of experiment-induced anxiety and thus the perceived stressfulness of the experimental situation (see Appendix B-6).During the experimental session subjective tension, stressfulness ("unpleasantness") of the situation and perceived effort to complete the given tasks were rated by the subjects, in addition to the perceived pain level (see 5.3.1.). The ratings ranged from 0 (not at all) to 10 (very much) (see Appendix B-4). The ratings were used to assess the immediate effects of the stress exposure on the subjects' experience of pain and tension. The unpleasantness rating of a situation was used as an indicator of the perceived stressfulness of the situation. The additional effort rating served the purpose of assessing the differences in mental strain between the tasks. Specifically, the mental arithmetic task was thought to be stressful due to the mental effort required.

Depression was assessed via Lubin's (1965, 1967, 1977) Depression Adjective Checklist (DACL) and Beck's (1967) Depression Inventory (BDI; see Appendix B-7 and B-8). The DACL is a measure of an individual's mood at a particular point in time, and may therefore be viewed as a measure of state depression which mainly assesses depressed affect. It consists of 34 adjectives that may be checked by an individual according to "...how you feel today-now...". The scale has high split half and parallel form reliability as well as high internal consistency (cf., Lubin, 1967; Lubin and Himelstein, 1976). A number of studies have demonstrated its validity (e.g., Christenfeld et al., 1978; Lubin, 1967, 1977) and norms are available (cf., Lubin & Levitt, 1979).

The BDI consists of 21 groups of items (with 4 - 6 items per group) assessing dysphoria, hopelessness, feelings of guilt, suicidal tendencies, tiredness, loss of appetite etc. They are coded from 0 to 3 with symptom severity increasing from 0 to 3. One item in each group has to be checked by the patient. A total score is computed from these items. The scale has sufficient reliability and validity and is a widely used measure of depression (cf., Beck, 1967; Forrest & Hokanson, 1975; Klein & Seligman, 1976).

Depression was not a central dependent variable but was rather thought to play a possible moderating role in the responsivity to stress.

Additional measures were:

(a) The Pain Experience Questionnaire (PEQ; Turk, Note 7) a brief, 4o-item likert-type scale evaluating the cognitive and emotional attributes of the pain experience (see Appendix B-9). Currently, its reliability and validity are still under investigation . However, for this study it was used to assess the amount of maladaptive feelings and thoughts about pain in the pain patients (i.e., appraisal, coping). It was expected to correlate positively with the physiological reactivity to stress.

(b) The Health Locus of Control Scale (HLC, Wallston et al., 1976; Wallston & Wallston, 1978; see appendix B-1o) is a measure of expectancy of reinforcement in health-related situations. It is an 18 -item likert-type instrument using 6-choice self-ratings ranging from "strongly disagree" to "strongly agree". Internal and external (with attribution of influence to powerful others or chance) locus of control scores can be computed. Reliability and validity of the scale are satisfactory (Lewis et al., 1978; Meyers et al., 1982; Tolor, 1978; Wallston

& Wallston, 1978).

Good copers were expected to have high internal control scores that would correlate negatively with stress responsivity, while bad copers would show high external scores and high reactivity to stress.

(c) The Locke Wallace Marital Adjustment Scale (MAS, Locke & Wallace, 1959; see Appendix B-11) is a wellknown measure of marital satisfaction with adequate reliability and validity (cf., Kimmel & Van der Veen, 1974; Locke & Wallace, 1959). The couples are asked to indicate seperately their general satisfaction with their marriage on a 7-point scale from 0 (very unhappy) to 35 (perfectly happy) as well as their agreement with their spouse (always agree to always disagree) within 8 areas of marriage (cf., sex relations, philosophy of life). Six more items address questions concerning the handling of disagreements to confidence in the mate. Marital (dis)satisfaction was viewed as a possible moderating variable. Marital discord may act as a potent stressor, marital satisfaction may attenuate aversive consequences of pain but may also be indicative of operant reinforcement of the pain problem.

5.5. Procedure

The experiment was conducted at the West Haven Veterans Administration Medical Center's psychophysiology laboratory from February 1982 to February 1983. At the beginning of the experimental session the subjects were informed about the purpose and the procedures of the study, and they were asked to read and sign the consent form. The wording of the verbal instructions was that of the written description of the study in the consent form (see Appendix C-1). Next the subjects were asked to fill out the state anxiety form and to answer questions on demographic and disease data (see Appendix B-4). Pain patients who had not gone through the PMP assessment were asked to fill out the McGill Pain Questionnaire, the Pain Experience Questionnaire and the Health Locus of Control Scales. Then the spinal mobility measure was taken. Subjects were instructed to "reach down to the floor with their fingertips, while keeping their knees straight, as far as they could." The fingertips-to-floor-distance was then recorded in centimeters.

After the subjects had washed their hands with soap (Ivory) provided by the experimenter, they were asked to sit in a straight-back arm chair

without leaning back, and with their hands and forearms placed on the arms of the chair and their feet on the floor.

Then the left and right back electrodes were attached after the skin had been cleansed with 70 % alcohol and rubbed with a mild abrasive, and after the electrodes had been filled with electrode cream and adhesive discs had been attached to them. The electrodes were attached at the described positions at the erector spinae or the trapezius muscles (see figure 2, page 42). Additional adhesive tape was used to keep the sensors firmly attached to the back. The patients were then allowed to lean back, and the frontalis electrodes were attached as described (see figure 2).

Next, the pletysmograph for the heartrate measurement was attached to the index finger of the subject's dominant hand, and the subjects were asked to let this hand hang down comfortably from the arm of the chair. The GSR sensors were attached to the hypo/thenar eminence of the nondominant hand after the sensors had been filled with surgical jelly.

A short test run of the equipment followed and any irregularities in electrode placement, the senor connections, or the modules (which had been calibrated prior to the session) were taken care of. Next, the patients were asked to sit still for 10 minutes with their eyes open and without moving, as soon as the signal was given (see Appendix C-2). They were asked to keep their eyes open (but without moving them much) in order simulate as closely as possible natural conditions in their environment. Before the signal for the beginning of the measurements was given, patients were asked to rate their pain and tension levels, to indicate the unpleasantness of the situation and the effort required to follow instructions at the moment, using a rating scale from 0 to 10 (see Appendix B-4). All the procedures added up to an adaptation period of approximately 15-20 minutes.

Then the 10-minute baseline recording phase began. The experimenter could control the physiological signals via a graphic display on the video screen. The subjects' view was shielded from the experimenter (who was in the same room) and the screen, as well as from the biolab modules. The investigator was, however, able to observe movements of the subject. The entire experiment took place in an approximately 3 x 2 m airconditioned and sound-attenuated lab. Although the room temperature

varied somewhat over the sessions these variations affected all groups equally.

After the 10-minute baseline, subjects were asked to repeat the pain, tension, unpleasantness and effort ratings. If necessary, they were allowed to move in the chair without getting up.

Then they were asked to assume their original position, the electrode placements were checked, and the subjects were instructed that the first experimental condition would follow. They were informed that the condition would consist of a 1-minute baseline and that they would then be instructed about the kind of task they were to perform (see Appendix C-3). Following the instructions a one-minute recording during the task performance would take place. After one minute of the task a beeper would sound, signalling them to stop their activity and a 5-minute final measurement would follow. They were asked to repeat back this sequence, and any questions they had were answered. Once again, they were instructed not to move during the recordings and to keep their eyes open. Then the 1-minute baseline was recorded. Following the baseline, the subjects received instructions for the task (see Appendix C-3). The tasks were given in a predetermined and counterbalanced order. The subjects were told to stop the task performance immediately on the beep-signal to allow for correct measurement of return to baseline.

At the end of the task (which was again signalled by a beeper), the patients rated again their pain and tension levels and the unpleasantness and effort of the task. The remaining tasks were given accordingly. Electrode placements and sensor connections were checked after each trial and corrected if necessary. After the presentation of the fourth task the electrodes were removed and the subjects could get up and move about. They were asked to fill out the State Anxiety Inventory once more. After a short debriefing the experimental session was terminated.

Apart from occasional equipment malfunction and loosening of the sensors due to movement between trials (which was easily corrected) few problems with the subjects occured. It was somewhat surprising how well the CBP patients could sit through the entire 90-minute session . Although they had to move beetween trials nobody asked that the session be terminated early (!).

6. Results

6.1. Data Reduction and Analysis

All physiological data were averaged for the baseline-period (10 minutes), the pretrial baseline (1 minute), the trial (1 minute) and the posttrial baseline (5 minutes). Return to baseline was calculated in seconds. It was defined as the point in time in the posttrial baseline when the physiological values first reached pretrial baseline values. A time of 300 seconds was recorded when the values did not return to pretrial baseline within the 5-minute posttrial interval and a value of 2 seconds (due to the 2-second averaging periods) was recorded when the values returned instantaneously to pretrial values. Those data were transferred to a UNIVAC 1100/80 computer for further analysis.

MANOVAs of the pretrial-trial differences in the physiological measures were computed (as suggested by Muller et al., 1983; Reinsel, 1982). MANOVA was preferred to repeated measures ANOVA because of the heterogeneous variances and heterogeneous variance-covariance matrices. Bartlett tests were significant at the .01 level for most physiological data. Geisser-Greenhouse (Geisser & Greenhouse, 1958) corrected repeated measures ANOVA was computed in comparison to MANOVA (cf., Keselman & Rogan, 1980) and yielded essentially comparable results. To test the experimental hypotheses specific a priori contrasts were used with mixed model ANOVA. MANOVA was also used instead of transformations because transformed values are less well interpretable. (M)ANOVA was preferred to a non-parametric solution (cf., Lienert, 1973) because it uses more information inherent in the data and contrast effects as well as interaction effects could be analyzed. (e.g., Overall & Klett, 1972).

Despite the criticisms of difference scores (cf., Cronbach and Furby 1971; Petermann, 1978) they were used in this analysis for several reasons. First, it is necessary to take baseline values into account when working with physiological data (see Wilder's (1950) formulation of the "law of initial values"; cf., Johnson and Lubin, 1972). The mere use of absolute values (cf., Collins et al., 1982) is not appropriate. In this study significant pretrial-trial correlations could be found in the physiological measures. ANCOVA with the baseline values as covariates seemed not advisable due to insufficient fulfilment of the

mathematical prerequisites (cf., homogeneity of variances and regression slopes). For the same reason multiple regressions - which are also less convenient to interpret - were not used. The decision to use change scores was also prompted by Cattell's (1983) recent defense of difference scores that delineates prerequisites of the reliability of change measures which are believed to be observed in this study.

In addition to these analyses univariate ANOVAs were computed for the physiological data when the assumptions of the statistical model were met; and they were also computed for a number of variables that differeentiated between the three groups (e.g., Keppel, 1982). Alternatively, Kruskal-Wallis ANOVAs were calculated if the data were not normally distributed and/or had unstable variances. T-tests or chi-square analyses were also computed for single (or multiple) group comparisons. Mann-Whitney U- and Wilcoxon tests were calculated for multiple group comparisons if the variables were not normally distributed and/or had unequal variances. In these cases probability levels that were corrected for the number of comparisons were used (cf., Kirk, 1968).

To analyze the relationship among the various physiological and psychological variables in and between the groups discriminant analyses as well as regression analyses and correlation analyses were performed. In the following section the results of these analyses will be presented for the physiological, motor- and the subjective levels of behavior.

6.2. Results on the Physiological Level

Hypotheses 1 to 7 in section 4.4. pertain to the physiological measures and results will be discussed seperately for each hypothesis.

6.2.1. Back Muscle Reactivity during Stress and Non-Stress Conditions

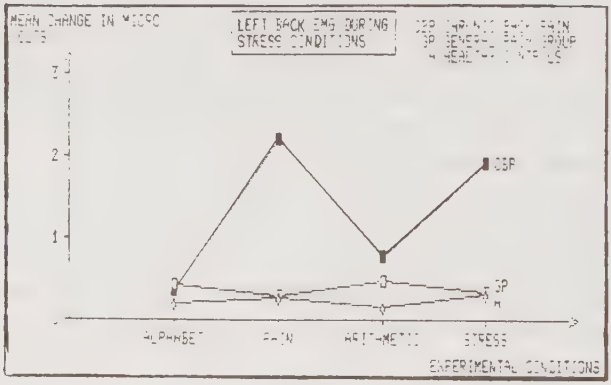
The reactivity of the subjects' left erector spinae muscles during the stress conditions is displayed in table 5 and figure 3. These show the mean change in EMG from pretrial baseline to trial for the three groups and four conditions.

TABLE 5

MEANS AND STANDARD DEVIATIONS OF THE LEFT BACK EMG CHANGES (IN MICRO-VOLTS) DURING STRESS EXPOSURE IN THE THREE GROUPS

	Alphabet M (SD)	Pain M (SD)	Arithmetic M (SD)	Stress M (SD)
Chronic Back Pain	.344 (1.043)	2.200 (2.645)	.768 (.879)	1.891 (2.181)
General Pain Group	.416 (.619)	.275 (.525)	.457 (.582)	.340 (.258)
Healthy Controls	.200 (.750)	.256 (.465)	.146 (.555)	.312 (.429)

FIGURE 3



Visual inspection of the data suggests an overreaction of the back muscles in the CBP group during the personally relevant stress situations (PAIN, STRESS) only. To test this hypothesis further the EMG changes were subjected to MANOVA, using the repeated measurements as response vector (cf., Nie et al.,1981). Contrasts comparing the CBP group with the two control groups and the two personally relevant stress conditions with the general stress and neutral situation yield the results that are displayed in Table 7 and the MANOVA results are shown in table 6.

TABLE 6

MANOVA FOR THE LEFT BACK EMG REACTION

Factor	Wilk's Lambda[1] or mean squares[2]	df	F	P
Groups	23.043[2]	2	8.839	.001
within	2.607[2]	48		
conditions	.858[1]	3,46	2.527	.069
Group by cond	.736[1]	6,92	2.536	.026

TABLE 7

CONTRASTS FOR THE LEFT BACK EMG REACTION

Factor	mean squares	df	F	P
Groups	181.533	1	17.408	.000
within	10.429	48		
Conditions	1.946	1	4.473	.040
Group by cond	3.424	1	7.870	.007
within	.435	48		

The MANOVA as a conservative estimate shows a highly significant group effect and a significant group by condition interaction. The hypothesis-specific a priori contrasts are all significant thus refuting the null hypothesis of no difference between the CBP and the two other groups on one hand, and the personally relevant versus the remaining conditions on the other h nd. The left back muscles react very strongly only in the CBP group and only in the personally relevant stress conditions, while the EMG changes in the other groups (H, GP) and conditions (mental arithmetic, alphabet) barely exceed the noise level.

Post hoc U-tests (adjusted alpha = .008) show significant differences only between the CBP group and the two other groups in the personal stress conditions, and Wilcoxon tests (adjusted alpha = .006) are significant only in the CBP group for the alphabet versus the pain and stress condition. The difference between the arithmetic and pain condition approaches significance ($p < .01$) only in the CBP group.

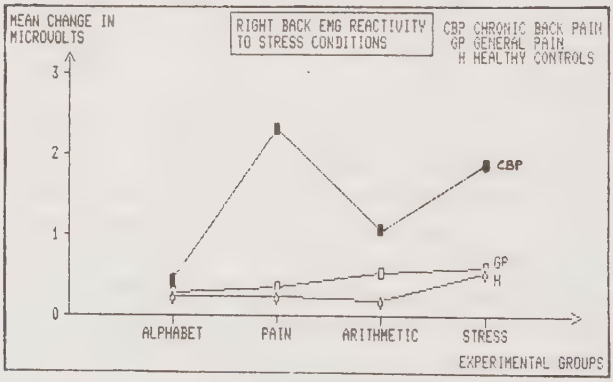
A similar effect can be demonstrated for the right back EMG values as table 8 and figure 4 show.

TABLE 8

MEANS AND STANDARD DEVIATION OF THE RIGHT BACK EMG CHANGES (IN MICRO-VOLTS) DURING STRESS EXPOSURE IN THE THREE GROUPS

	Alphabet M (SD)	Pain M (SD)	Arithmetic M (SD)	Stress M (SD)
Chronic Back Pain	.415 (.639)	2.297 (2.611)	1.046 (1.342)	1.737 (1.660)
General Pain	.269 (.317)	.325 (.406)	.512 (.751)	.584 (.348)
Healthy Controls	.210 (.359)	.222 (.265)	.166 (.596)	.509 (.400)

FIGURE 4



The effect for the right back is similar to that of the left back: the CBP group alone shows abnormal EMG activity only in the personally relevant stress conditions (PAIN, STRESS) but not in the general stress (arithmetic) and neutral (alphabet) condition. Tables 9 und 1o show the results of the MANOVA and ANOVA with a priori contrasts for the right back EMG changes.

TABLE 9

MANOVA FOR THE RIGHT BACK EMG REACTION

Factor	Wilk's lambda[1] or mean squares[2]	df	F	P
Groups	27.725[2]	2	11.622	.000
within	2.385[2]	48		
conditions	.782[1]	3.46	4.277	.010
Group by Conditions	.651[1]	6.92	3.667	.003

TABLE 10

CONTRASTS FOR THE RIGHT BACK EMG CHANGE

Factor	mean squares	df	F	P
groups	226.212	1	22.912	.000
within	9.873	48		
conditions	2.391	1	7.362	.009
group by cond.	3.344	1	10.295	.002
within	0.325	48		

The MANOVA shows significant group and condition effects and a highly significant interaction, thus refuting the null hypothesis. The contrasts all yield very significant results and therefore indicate a differential effect for the CBP group versus the control groups, and the personally relevant stress situations versus the other two conditions. The data fully support the prediction of hypothesis 1 for the right and for the left back EMG changes. Post-hoc U- and Wilcoxon tests were significant for the predicted group ($p = .008$; CBP versus GP, H in the personal stress conditions) and condition ($p = .006$; alphabet versus pain, stress; arithmetic versus stress in the CBP group only) differences.

The described response can also be seen in the absolute EMG values. Figures 5 and 6 display the pretrial, trial, and posttrial absolute EMG values for the four experimental conditions. The means and standard deviations are listed in appendix D-1.

FIGURE 5

LEFT BACK ABSOLUTE EMG LEVELS PRE, DURING AND POST STRESS EXPOSURE

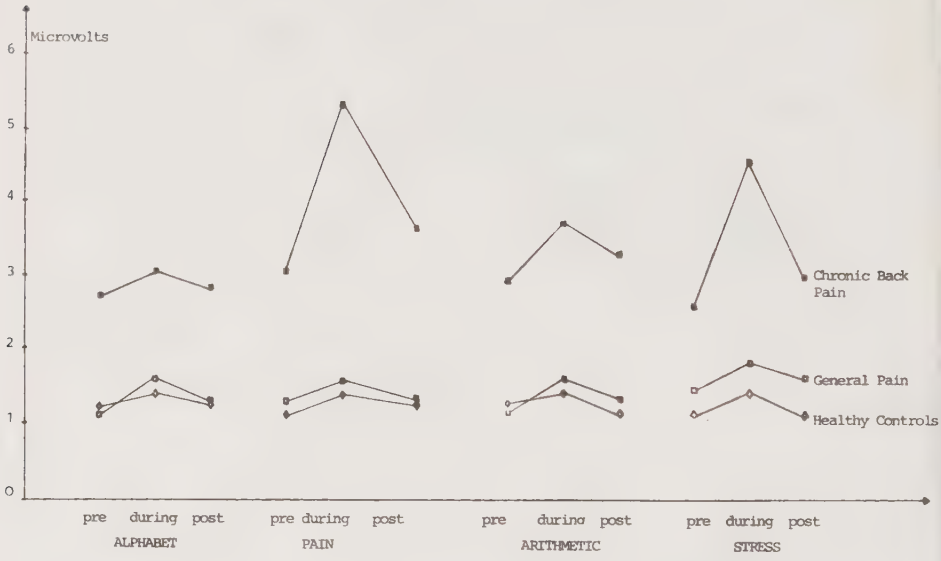
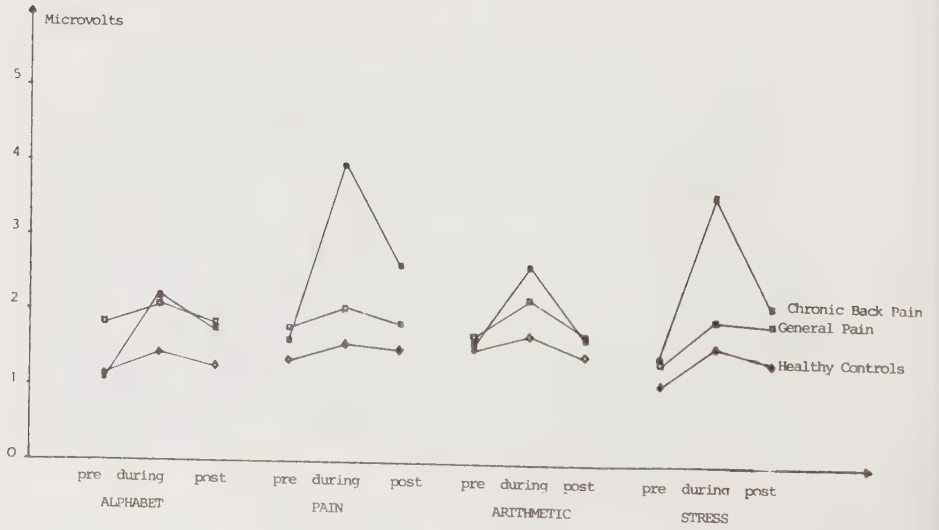


FIGURE 6

RIGHT BACK ABSOLUTE EMG LEVELS PRE, DURING AND POST STRESS EXPOSRUE



An additional question had been if the EMG reactivity would be especially dramatic at the affected side of the back if the pain was unilateral. Examination of the patient interviews shows that more than 5o % of the patients in the pain groups do have a dominant unilateral pain site. Table 11 shows the distribution of the laterality of the pain.

TABLE 11

LATERALIZATION OF THE PAIN IN THE TWO PAIN GROUPS

	Left N	Right N	Both sides N
Chronic Back Pain	9	5	3
General Pain	3	6	8

The chronic back pain group shows a slight dominance of the left side versus the right (Binomial test : p = .122), while the general pain group has a dominance of bilateral or right-sided pain. To test the hypothesis of side-specific reactivity the subjects' left back EMG was used when the pain was localized on the left side, the right back EMG was used when the dominant pain site was at the right side, and the average of both sides was used if no side-dominance existed, and also for the healthy controls. Then a new MANOVA of the EMG change using those values was computed. Table 12 shows the means and standard deviations of the combined variable and table 13 displays the results of the MANOVA.

TABLE 12

MEANS AND STANDARD DEVIATIONS OF THE SIDE-SPECIFIC BACK EMG CHANGE (IN MICROVOLTS)

	Alphabet M (SD)	Pain M (SD)	Arithmetic M (SD)	Stress M (SD)
Chronic Back Pain	.379(1.121)	2.185(2.757)	1.079(1.325)	2.163(2.270)
General Pain	.346(.413)	.301(.358)	.543(.740)	.479(.303)
Healthy Controls	.205(.328)	.239(.333)	.156(.540)	.410(.387)

TABLE 13

MANOVA FOR THE BACK EMG CHANGE AT THE RELEVANT SIDE

Factor	Wilk's lambda[1] or mean squares[2]	df	F	p
Groups	28.708[2]	2	8.603	.001
within	3.337[2]	48		
conditions	.769[1]	3,46	4.597	.007
Group by Cond.	.711[1]	6,92	2.846	.014

Comparison of these site-specific EMG changes to those of the right and left back shows that the paravertebral EMG levels of the CBP groups do partially increase when the reactivity of the pain-specific side is used as a dependent measure. The MANOVA, too, has a tendency to more significant values as compared to those of the right and left back values only; this may also be due to the lower EMG-levels of the control groups when the pain-site specific values are chosen.

To summarize, the null hypothesis of no difference in the back EMG response levels to stress between the groups and the conditions must be rejected. The data show a clear differential effect in EMG reactivity for the CBP patients in the personally relevant stress conditions for the left as well as for the right back musculature, or the pain-relevant side alone.

6.2.2. Return to Baseline of the Back EMG Values after the Stress and Non-Stress Conditions

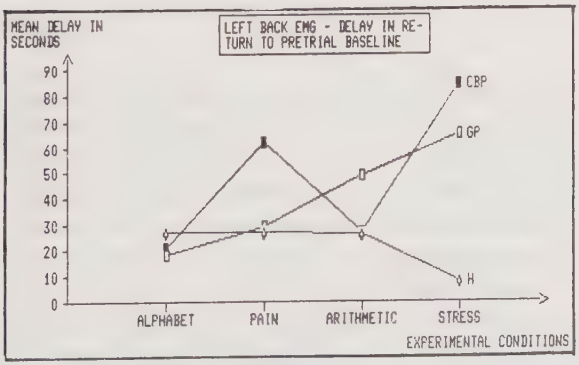
Hypothesis 2 predicted delayed return to pretrial baseline values of both the right and left back EMG values in the CBP group following exposure to personally relevant stress. Table 14 and figure 7 show the return to baseline for the left back EMG.

TABLE 14

MEANS AND STANDARD DEVIATIONS FOR THE DELAY IN RETURN TO BASELINE (IN SECONDS) OF THE LEFT BACK EMG VALUES

	Alphabet M (SD)	Pain M (SD)	Arithmetic M (SD)	Stress M (SD)
Chronic Back Pain	2o.59(44.83)	62.oo(87.oo)	26.65(7o.93)	85.29(99.4o)
General Pain	18.35(52.6o)	29.53(71.3o)	49.18(95.77)	65.18(113.6o)
Healthy Controls	27.06(71.48)	27.94(71.52)	26.oo(65.33)	8.12(71.24)

FIGURE 7



The differential effect as found in the EMG reactivity is less pronounced in the delay to return to baseline, but nevertheless still visible. The CBP group data clearly show long delays in the return to baseline during the pain and stress conditions as compared to the neutral and arithmetic conditions. The EMGs of the healthy controls show minimal delay in return to baseline across all conditions. In the general pain group an increased delay can be found in the general stress and the personal stress conditions, though this delay remains below that of the CBP group (except for the arithmetic condition). The results of the two-factorial ANOVA with repeated measures on the factor time utilizing contrasts for groups and conditions are displayed in table 15.

TABLE 15

ANOVA (CONTRASTS) FOR THE TIME TO RETURN TO BASELINE OF THE LEFT BACK EMG VALUES

Factor	mean squares	df	F	p
Groups	5770o.74	1	1.2o7	.277
within	47818.73	48		
Conditions	4564.83	1	3.431	.o7o
Group by Cond.	1oo6o.48	1	7.562	.oo8
within	133o.49	48		

The analysis shows a non-significant group effect and an almost significant condition effect, but a highly significant interaction. The return to baseline measure thus also differentiates the CBP group from the other groups in its reaction to the personally relevant versus the other stress conditions. Single group or condition comparisons confirm this result. The CBP group and the healthy control group differ significantly from each other in the pain and stress conditions ($p = .oo8$). The alphabet condition differs significantly from the pain and stress condition, and the arithmetic condition differs significantly from the stress condition only in the CBP group, and the alphabet differs from the arithmetic and stress condition in the GP group (all $p < .oo6$).

The return to baseline of the EMG values from the right side of the

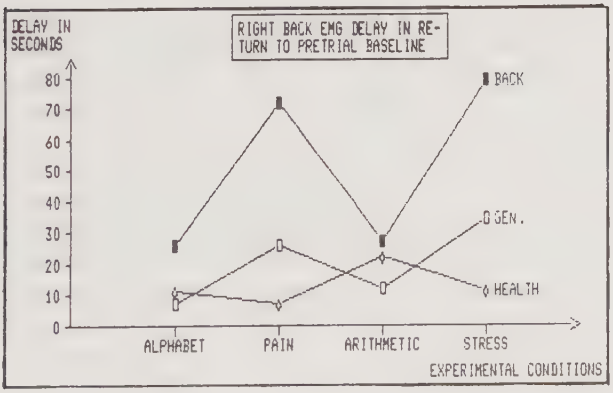
of the back shows a similar pattern. Table 16 and figure 8 display these values.

TABLE 16

MEANS AND STANDARD DEVIATIONS OF THE TIME TO RETURN TO BASELINE (IN SECONDS) FOR THE RIGHT BACK EMG

	Alphabet M (SD)	Pain M (SD)	Arithmetic M (SD)	Stress M (SD)
Chronic Back Pain	25.59(44.83)	71.53(113.5o)	26.95(32.o9)	79.41(1o2.94)
General Pain	18.35(52.6o)	26.12(7o.99)	11.77(13.54)	34.23(76.82)
Healthy Controls	27.o6(71.48)	6.59(16.75)	22.12(59.59)	11.o6(16.17)

FIGURE 8



Here, too, the healthy control group shows only minimal delay in return to baseline, with no obvious condition differences, the GP group shows somewhat longer delays, and only the CBP group shows clear increase in delay in the personal stress conditions. Table 17 presents the ANOVA data.

TABLE 17

ANOVA (CONTRASTS) FOR THE TIME TO RETURN TO BASELINE OF THE RIGHT BACK EMG VALUES

Factor	mean square	df	F	p
Groups	218415.686	1	12.314	.001
within	17737.392	48		
conditions	5781.352	1	3.315	.075
Group by Cond.	6196.323	1	3.552	.066
within	1744.091	48		

The ANOVA reveals a very significant group effect, and close to significant condition and interaction effects. There is a strong tendency for a differential effect of the return to baseline for the right back EMG levels, i.e. the CBP group reacts with more delay to the pain and stress conditions. This tendency is confirmed by the post hoc comparisons. With alpha set at .008, the CBP group and the healthy control group differ significantly in the pain and stress situation. The differences between the GP and H, and the CBP and GP group are not significant at the predetermined alpha level. Within-group comparisons are significant only in the CBP group. The alphabet condition differs from the stress condition below the preset alpha-level of .006.

Analysis of the delay in return to baseline at the pain-specific side of the body yields results comparable to those of both sides, except for a more significant interaction as compared to the right back values alone ($F_{1,48} = 7.025$; $p = .011$). The delay, however, is not uniformly prolonged at the relevant side as compared to the other sides measured seperately (see table 18).

TABLE 18

TIME TO RETURN TO BASELINE (IN SECONDS) OF THE PARASPINAL EMG AT THE PAIN-RELEVANT SIDE

	Alphabet M (SD)	Pain M (SD)	Arithmetic M (SD)	Stress M (SD)
Chronic Back Pain	25.118(57.834)	5o.411(75.627)	13.588(12.679)	79.ooo(94.61o)
General Pain	12.294(32.478)	18.529(35.779)	21.ooo(36.2o3)	54.412(99.392)
Healthy Controls	18.941(36.6o9)	14.765(35.543)	24.o59(61.937)	9.588(1o.96o)

To summarize the results for hypothesis 2: The delay in return to baseline of the back EMGs is similar although less significant than the increases in EMG level. The left back displays a clear interaction effect for the CBP versus the two other groups and the personally relevant stress conditions versus the other two conditions; the right back return to baseline shows a close to significant interaction effect. The null hypothesis of no differences in time to return to baseline for the back EMG in the various stress conditions must clearly be refuted for the left back and the affected side, probably also for the right back.

6.3. Resting Baseline EMG Levels at the right and left Back

The subjects' right and left paraspinal resting EMG levels were measured during a 1o-minute baseline that followed the initial 15-minute adaptation phase. Table 19 shows the subjects' mean left and right paraspinal EMG levels during this 1o-minute sitting period. Medians and Kruskal-Wallis ANOVA are also displayed. They were computed with regard to the large variance of the left back EMG level in the CBP group (range .35 to 28.8 microvolts). Figures 9a and 9b show the left and right back EMG resting levels for the three groups.

TABLE 19

LEFT AND RIGHT MEAN AND MEDIAN PARAVERTEBRAL EMG LEVELS (SITTING) IN MICROVOLTS AND ANOVA FOR THE GROUPS

	left back			right back		
	M	(SD)	Mdn	M	(SD)	Mdn
Chronic Back Pain	3.626	(6.795)	1.400	2.479	(2.210)	1.625
General Pain	1.326	(1.206)	.900	1.565	(.980)	1.175
Healthy Controls	1.227	(.633)	1.100	1.469	(.880)	1.281
Kruskal-Wallis ANOVA:	X^2 = 6.327		p=.042	X^2 = 2.783		p = .249

FIGURE 9a

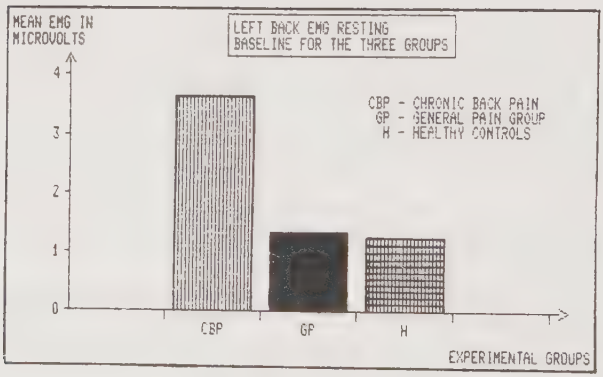
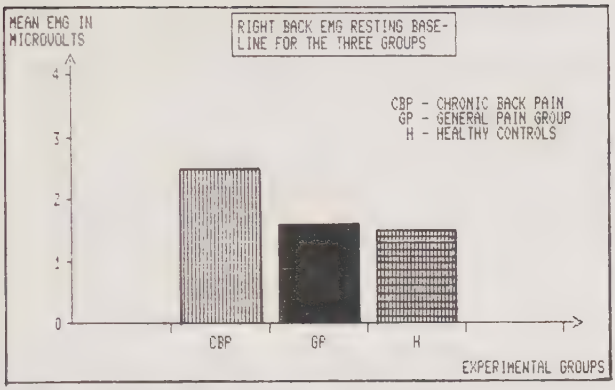


FIGURE 9b



The resting tension levels in the three groups do not differ as dramatically as the EMG changes during stress. While the EMG values at the left side of the back differ significantly at the .042 level (with the CBP being significantly different from the GP group ($p < .05$), the right back EMG levels are not significantly different. Both sides of the back do, however, display the highest resting tension levels in the CBP group as compared to the two other groups. The absolute levels are not very high, however, the values were recorded during relaxed sitting. Interesting is the high variance in the CBP group that is due to the extremely high EMG levels of only a few CBP patients, while the majority have moderately high EMG levels as the medians show. Due to this large variance difference Kruskal-Wallis ANOVA was computed. To assess the hypothesis of higher resting tension levels at the affected side of the back, the mean and median EMG for only the affected side of the back was computed, and another Kruskal-Wallis ANOVA was performed. Table 2o shows the results of this analysis.

TABLE 2o

MEANS, STANDARD DEVIATIONS AND ANOVA FOR THE BACK EMG RESTING LEVEL AT THE AFFECTED SIDE

	M	SD	Mdn	
Chronic Back Pain	3.524	6.727	1.6o	$x^2 = 12.593$
General Pain	1.439	.96o	1.15	
Healthy Controls	1.348	.695	1.3o	p = .oo2

Table 2o shows that the absolute EMG level is higher than that of the right back when the relevant side-EMG is used as compared to assessing the EMGs of both sides of the back separately. The CBP group differs significantly from the GP group (p = .o44) and almost significantly from the healthy controls (p = .o56) . In comparison to the control groups - whose EMGs are somewhat lowered in this procedure - this difference becomes more significant than the original unilateral EMGs. The hypothesis of a still more elevated EMG at the pain-relevant side of the back could thus partly be established. In general, there seems to be a tendency for rather stronger reactions from the left side of the back, though direct comparison of the two sides does not yield significant differences.

Following Fassbender (e.g., 1975) it was hypothesized that with increasing duration of the pain problem the back tension levels might be lowered due to muscle fiber destruction. The correlation coefficient between the left back resting EMG level and the duration of the pain problem is -.2162 (p = .211) and for the right back EMG level -.2918 (p = .136). Although the correlation points in the predicted direction, this correlation does not become significant, nor can U-tests differentiate between the pain duration of the high and low EMG groups (median split; left: z = -.2315; p = .817; right: z = -1.217, p = .224). To summarize, the paraspinal resting baseline tension levels are higher in CBP patients than in general pain patients or in healthy controls, although this difference is statistically significant only for the left side of the back or when the EMG at the affected side is evaluated. However, this difference is not very dramatic, and the CBP patients' absolute EMG levels are

still rather low. The levels do not show a significant relationship to the duration of the pain problem, although the correlation between these two variables is negative. Basmajian's (1976) assertion that EMG levels inspastic muscles cannot be assessed via surface electrodes could neither be refuted nor confirmed. More basic research to address this question is needed. The suggestion of Wolf and his collegues (1976, 1982) that side asymmetries per se may be more decisive than high tension levels could also not be confirmed. Only few asymmetries were noted (only 6 patients in the CBP group had side differences of more than 1 microvolt and only 3 more than 2 microvolts). Although a significant relationship was found between the relevant pain site (left, right or both sides of the back) and baseline muscle tension, as well as muscular reactivity to stress, the muscular reactions were not much more pronounced at the affected side as compared to the left side (that was slightly more affected) or the right side alone. There is no relationship between side asymmetry and amount of pain (correlation of the amount of asymmetry with the pain card index:r = .o76; with the McGill total score: -.o97.) However, since the measurements were only taken during sitting, it cannot be excluded that stronger side asymmetries may be present in other positions or during movement (cf., Wolf et al., 1982).

6.2.4. Relationship of the Back EMG Activity to Organic and Psychological Variables

It was hypothesized that the EMG reactivity of the back may be more clearly related to the ability to cope with stress and to present anxiety and immobility levels (see hypotheses 8 - 1o) than to organic variables such as the amount of degenerative changes present in the spine, any previous back surgeris or the number of previous back surgeries. A median split of the patients in the CBP group into those with high versus low paraspinal EMG levels and a subsequent crosstabulation with the amount of degenerative change was performed. Table 21 shows this crosstabulation and the chi-square analysis.

TABLE 21

HEIGHT OF BACK EMG BASELINE LEVELS BY AMOUNT OF DEGENERATIVE CHANGE (X-RAYS) IN THE CBP GROUP

	Low EMG Level	High EMG Level
X-Rays:		
No Change	4	3
Slight Change	1	o
Moderate Change	1	4
Severe Change	2	1

$X^2 = 4.232$ $p = .3755$

The X^2 analysis does not show a relationship between the EMG baseline levels in the back and the amount of degenerative change as determined by x-rays. Nor is there a significant relationship between back surgery and EMG levels or the number of back surgeries and EMG levels as table 22 shows.

TABLE 22

BACK SURGERY AND NUMBER OF SURGERIES BY HEIGHT OF PARASPINAL EMG LEVELS FOR THE CBP GROUP

		BACK EMG LEVEL Low	BACK EMG LEVEL High	Significance Test
Surgery	Yes	5	4	Fisher's exact test
	No	3	5	(1-tailed) p = .3992
Number of Surgeries	o	5	4	
	1	2	1	$X^2_{(3)} = 3.397$
	2	o	3	
	3	1	1	p = .334

The back EMG levels were instead significantly related to the amount of anxiety (STAI for the low level group: M = 38.oo, SD = 7.5o; STAI for the high tension group: M = 47.33, SD = 9.421; $t_{(15)}$ = 2.238, p(1-tailed = .o2o), the state depression as measured with the DACL (DACL for the high EMG group : M = 14.44, SD = 5.57o; DACL for the low EMG group: M = 1o.oo, SD = 1.69o, $t_{(15)}$ = 2.163, p(1-tailed) = .o24) and the subjective effort rating before the experiment (effort for the low EMG group: M = 2.375, SD = 2.925; effort for the high EMG group: M = 5.889, SD = 3.4o8; $t_{(15)}$ = 2.267, p = .o2o). Stepwise regression analysis shows that the resting EMG levels of the back can best be predicted by the McGill evaluative dimension (ß = -.145),the PEQ score (helplessness, lack of coping ability, ß = .742), the immobility measure (ß = .374), and the Beck Depression Score (ß = -.613; R = .898, R^2 = .8o6, $F_{4,8}$ = 8.331, p < .o1).

Similar results can be found for the EMG reactivity. The CBP patients were median split into two groups according to the reliability of their assignment to the CBP group versus the other two groups on grounds of their back EMG reaction to the personally relevant stressors in a discriminant analysis procedure (see 6.2.5). While 9 patients showed moderate to high reactivity levels ("responders"), 8 patients displayed only low to moderate ("nonresponders") paravertebral EMG reactions to the personally relevant stress situations. Crosstabulation of responders versus nonresponders with the variables amount of degenerative change, surgery, number of surgeries and duration of the pain and subsequent chi-square , Fisher's exact test or t-test analysis, did not yield any significant differences between these two groups (see table 23).

TABLE 23

CROSSTABULATION AND ANALYSIS OF THE BACK EMG RESPONDERS VS. NON-RESPONDERS BY SURGERY, NUMBER OF SURGERIES, DEGENERATIVE CHANGE, DURATION OF PAIN

		Responders	Nonresponders	Significance Test
Surgery	Yes	4	4	Fisher's exact test:
	No	5	4	p = .601
Number of Surgeries	0	5	4	
	1	1	2	X^2_3 = .721
	2	2	1	
	3	1	1	p = .868
Amount of Degenerative Change (x-rays)	none 0	4	3	
	slight 1	0	1	X^2_3 = 1.449
	moderate 2	3	2	
	severe 3	2	1	p = .694
Pain Duration (in years)	M	13 yrs 3 mths	9 yrs 10mths	$t_{(14)}$ = .500
	SD	13 yrs 8 mths	13 yrs	p = .623

Analysis of the psychological variables shows significant differences for anxiety (STAI-responders : M = 46.556, SD = 10.345; nonresponders: M = 38.875, SD = 7.240, $t_{(15)}$ = 1.750 p (1-tailed) = .050), depressed mood (DACL negative adjectives - responders: M = 6.000, SD = 4.664; nonresponders: M = 2.125, SD = 2.475; $t_{(15)}$ = 2.10, p(1-tailed) = .027), and trends towards significant differences for marital satisfaction (MAS - responders : M = 109.167, SD = 14.716; nonresponders: M = 120.500, SD = 14.571, $t_{(10)}$ = -1.340, p(1-tailed) = .100), the DACL total score (responders: M = 13.778, SD = 5.495; nonresponders: M = 10.750, SD = 3.196; $t_{(15)}$ = 1.360 p = .097) and general mental status (status (ranging from 0 = not affected to 3 = very affected : responders M = 1.111, SD = .782; nonresponders: M = .400, SD = .894; $t_{(12)}$ = 1.55, p(1-tailed) = .073).

To summarize, the responders are more anxious, more depressed, tend to be less satisfied with their marriage, and tend to be more psychologically affected, than the non-responders. These variables may be instrumental for this group's muscular stress reactions.

6.2.5. Classification of the Subjects into the Three Experimental Groups and Back EMG Changes During Treatment

Hypothesis 5 suggests that the subjects can be assigned to the three groups - chronic back pain, other pain, or healthy controls - on grounds of their back EMG reactivity. Specifically it is expected that the CBP patients can be reliably assigned to the CPB group versus the general pain and the healthy control groups. To test this hypothesis a discriminant analysis was performed. The variables entered into the analysis were the left and right back EMG reactions to the 4 experimental conditions. Stepwise discriminant analysis was first performed for differentiation of all threee groups to evaluate the assumption that although the variables could differentiate the CBP from the GP group and the healthy control group, the two control groups could not be differentiated in their back EMG reaction - thus suggesting a back pain specific effect. Then a discriminant analysis differentiating the CBP group from the other two groups together was performed. The goal of both analyses was to determine if the groups could be reliably differentiated by their back muscle reactions. Tables 24 and 25 show the results of the discriminant analyses.

TABLE 24a

RESULTS OF THE DISCRIMINANT ANALYSIS FOR THE BACK REACTIVITY MEASURES IN THE CBP GROUP VERSUS THE TWO CONTROL GROUPS

Function	% Variance Explained	Canonical Correlation	Wilk's Lambda	chi^2	df	p
1	92.12	.647	.548	27.654	1o	.oo2
2	7.88	.241	.942	2.743	4	.6o2

Function Variables	Standardized Coefficients	
Function	1	2
Right Back EMG-Stress	1.183	-.119
Left Back EMG-Pain	1.o25	-.582
Left Back EMG- Stress	-.953	.426
Left Back EMG-Arithmetic	-.o71	.9344
Left Back EMG Alphabet	-.515	.567

		Predicted Group Membership		
		CBP	GP	H
Actual Group Membership	CBP	11 (64.7%)	3(17.6%)	3(17.6%)
	GP	o (o %)	6(35.3%)	11(64.7%)
	H	1 (5.9%)	2(11.8%)	14(82.3%)

TABLE 24b

RESULTS OF THE DISCRIMINANT ANALYSIS FOR THE BACK REACTIVITY MEASURES IN THE CBP GROUP VERSUS THE NON-CBP CONTROLS

Function	% Variance Explained	Canonical Correlation	Wilk's Lambda	chi^2	df	p
1	1oo	.666	.556	22.867	4	.ooo1

Function Variables	Standardized Coefficients
	Function 1
Left Back EMG-Stress	.9423
Right Back EMG-Pain	.7o89
Right Back EMG-Stress	.49o9
Left Back EMG-Arithmetic	.2934

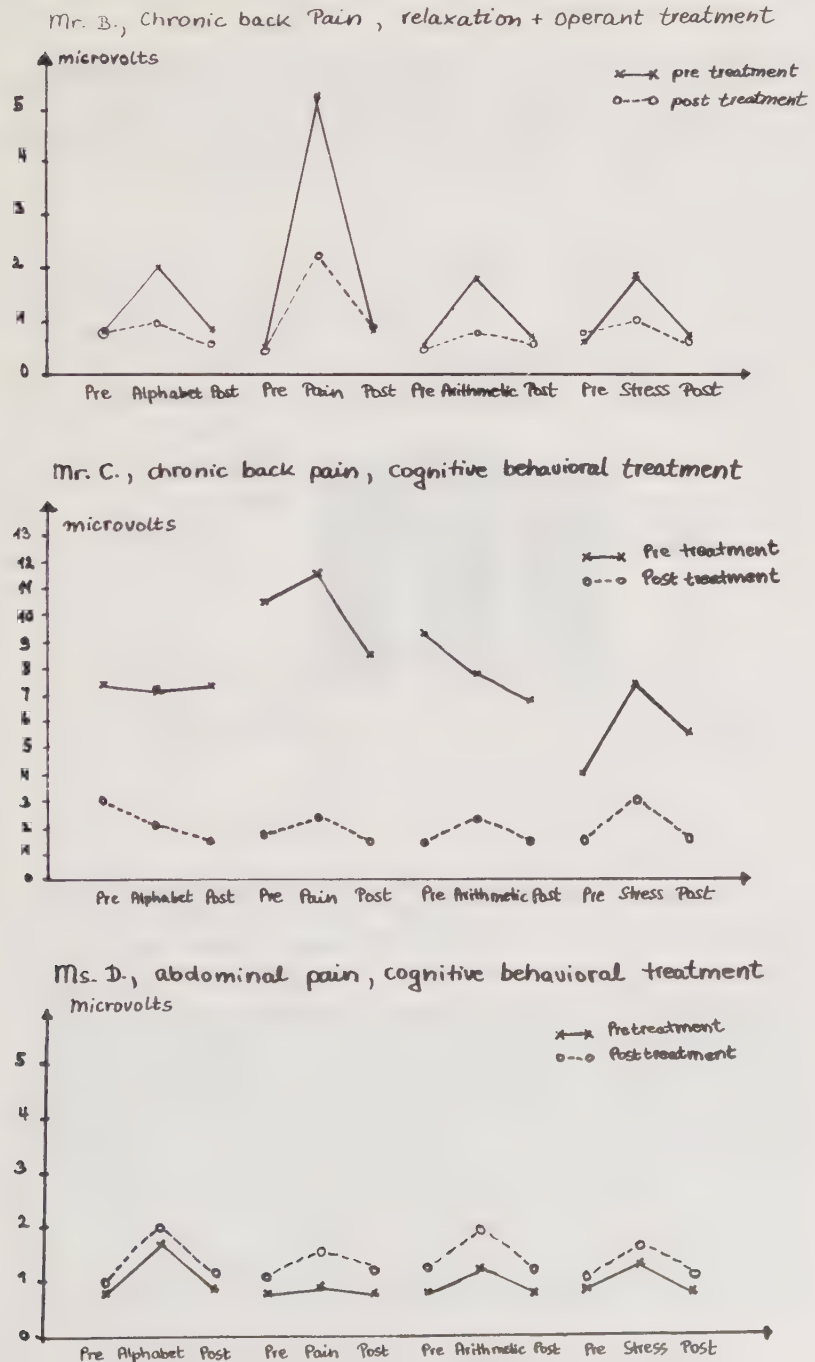
		Predicted Group Membership	
		CBP	NON-CBP
Actual Group Membership	CBP	13(76.5%)	4(23.5%)
	NON-CBP	1(2.9%)	33(97.1%)

The analysis shows that the reactivity measures fairly reliably classify the back pain patients versus the general pain and healthy control groups, but do not differentiate as well between the two latter groups. The percentage of correct classifications is 6o.78% (a priori 33.3%), when all three groups are considered, and it is 9o.2% (a priori 5o%) if the classification is limited to the CBP group versus the two controls. The majority of the values of the general pain group resembles those of the healthy controls - as had been predicted. Only 1 patient in the healthy control group, and none of the patients in the general pain group were mistakenly placed in the CBP group. The discriminant analysis for the CBP versus the healthy controls shows that the majority of the CBP patients can be reliably classified into the CBP group (76.5%). However, 23 % of the CBP patients fall into the other groups, i.e., they do not show abnormal back muscle reactivity. The question of why this may be the case will be discussed in more detail in section 6.4. (see also 6.2.3.).

A further assumption had been that psychological treatment teaching stress-management (be it via relaxation or cognitive coping sills training, for example) would attenuate the back muscle reactivity during stress exposure. Although some of the pain patients in this study underwent 1o weeks of either cognitive-behavioral treatment or relaxation plus operant treatment, no systematic control group data can be presented here because only a few patients returned for a second post-treatment psychophysiological session. The anecdotal evidence available, is, however, promising. Figure 1o displays the pre- and posttreatment left back EMG reactions (they were most pronounced) during the four experimental conditions for two chronic back pain patients and a patient with abdominal pain.All those patients had experienced more than 5o % pain reduction during the course of the treatment and could be considered successful participants of the program. The data show that both chronic back pain patients, patient B. and patient C., displayed abnormal reactions in their back muscles to the pain and/or stress situation in the pretreatment session, with patient C. showing additionally abnormally high EMG baseline levels and EMG variability. Patient D., who suffered from abdominal pain, did not display unusual back muscle reactions at pretreatment. At the post treatment measurement the back EMG reactions of both CBP patients were considerably attenuated, with patient C. also displaying much lower baseline levels. The GP patient (D) showed similar reactivity as during the pretreatment session, with the resting levels minimally higher. This essentially equivalent reaction during retest suggests that the CBP groups' lower reactions are not only due to habituation. Although these data cannot be considered as evidence, they suggest that abnormal physiological activity in the back muscles of CBP patients may be attenuated by psychological treatment, and this reduction in EMG reactivity may be instrumental for the pain reduction occuring during treatment. This hypothesis should be investigated more fully in subsequent research.

FIGURE 10

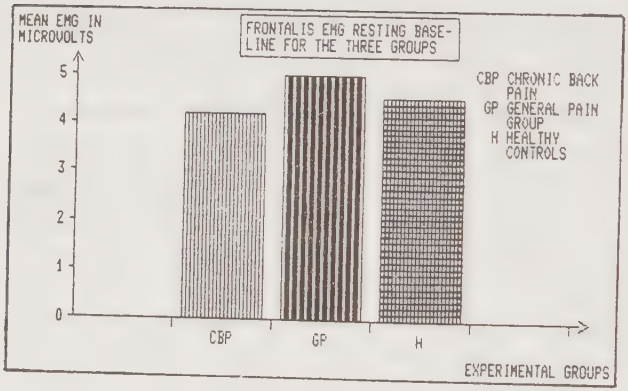
PRE- AND POSTTREATMENT LEFT PARASPINAL MUSCLE TENSION LEVELS OF TWO BACK PAIN AND ONE ABDOMINAL PAIN PATIENT DURING THE EXPERIMENTAL CONDITIONS


Mr. B., Chronic back Pain, relaxation + operant treatment
microvolts
pre treatment
post treatment
Pre Alphabet Post Pre Pain Post Pre Arithmetic Post Pre Stress Post
Mr. C., chronic back pain, cognitive behavioral treatment
microvolts
Pre treatment
Post treatment
Pre Alphabet Post Pre Pain Post Pre Arithmetic Post Pre Stress Post
Ms. D., abdominal pain, cognitive behavioral treatment
Microvolts
Pretreatment
Post treatment
Pre Alphabet Post Pre Pain Post Pre Arithmetic Post Pre Stress Post

6.2.5. Frontalis Muscle Activity During Baseline and Stress Conditions

The resting baseline tension levels for the frontalis muscle are displayed in figure 11 (for means and standard deviations, see Appendix D-2).

FIGURE 11



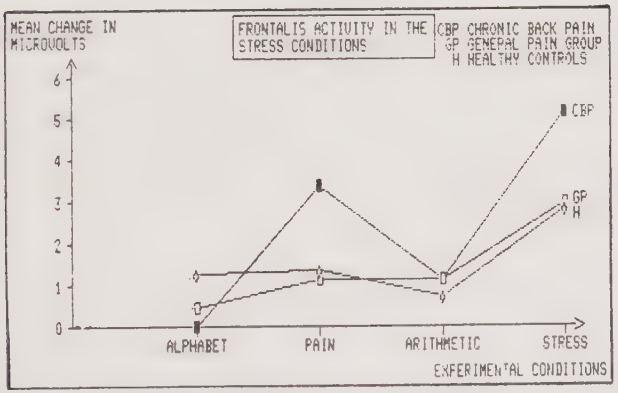
The three groups do not differ significantly in forehead tension (ANOVA: $F(2,48) = .4o3$, $p = .67o$). Hypothesis 6 had postulated that all groups would show marked frontalis EMG response during mental arithmetic, with the pain groups displaying a stronger reaction than the healthy controls. It was also hypothesized that there would be a (less strong) frontalis reaction in the pain and stress condition which would be more pronounced in the pain groups than in the healthy controls. Table 25 and figure 12 show the mean EMG change of the frontalis muscle during the stress conditions.

TABLE 25

MEAN FRONTALIS EMG CHANGE (IN MICROVOLTS) DURING STRESS EXPOSURE

	Alphabet M	Pain M	Arithmetic M	Stress M
Chronic Back Pain	.079 (2.75c)	3.362 (5.773)	1.135 (2.816)	5.104 (6.287)
General Pain	.446 (3.446)	1.109 (2.268)	1.129 (4.735)	2.916 (4.687)
Healthy Controls	1.259 (2.384)	1.350 (2.371)	.716 (3.088)	2.781 (4.019)

FIGURE 12



The data suggest that the CBP group shows an EMG reaction in the frontalis muscle that is comparable to that of the back muscles, although much less pronounced. The pain and stress situations show the strongest reactions in the CBP group. The other two groups show similar (low) responses in all but the stress condition, which elicited a somewhat higher reaction. The results of the MANOVA and contrasts comparing the arithmetic condition with the other conditions (contrast 1), or all stress conditions to the neutral condition (contrast 2), and the pain groups with the the healthy controls (as was suggested in hypothesis 6) are displayed

in table 26.

TABLE 26

MANOVA AND CONTRASTS FOR THE FRONTALIS STRESS REACTIONS

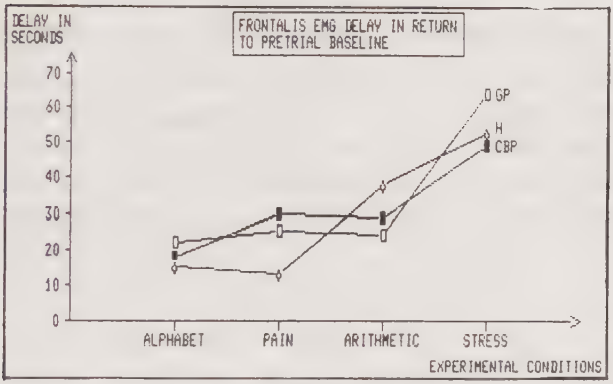
Factor	Wilk's Lambda (1) or Mean Square (2)	df	F	p
Groups	19.344 (2)	2	.584	.561
within	33.097 (2)	48		
Conditions	.643 (1)	3,46	8.502	.000
GroupxCond.	.872 (1)	6,92	1.090	.172
Groups (Contrast)	5.281 (2)	1	.048	.827
within	108.776 (2)	48		
Conditions (Contrast 1)	1.220	1	1.504	.226
Cond. (Contrast 1) x Group (Contrast)	.352	1	.433	.513
within	.812	48		
Conditions (Contrast 2)	8.876	1	8.995	.004
Cond. (Contrast2) x Group (Contrast)	.019	1	.011	.917
within	.977	48		

The results only partially support the assumptions of hypothesis 6. There is no stimulus-specific fontalis EMG reaction to mental arithemtic in any group. The specific contrast for this hypothesis is not significant ($p = .226$ for contrast 1), while the MANOVA yielded a significant condition effect in general. The hypotehsis of a stronger reaction of the frontalis muscle during the stress conditions versus the neutral condition could, however, be supported ($p = .004$ for contrast 2). The assumption of a stronger reaction of the pain versus the control group could not be supported ($p = .513$ or $.917$, respectively). U-tests do not differentiate the three groups in their frontalis reactivity. Wilcoxon tests yield significant differences between the stress and the alphabet and pain condition, the alphabet and pain condition and the arithemtic and pain condition in the CBP group (alpha = .006), between the alphabet and stress condition in the general pain group, and for the alphabet and arithmetic condition

versus the stress condition in the healthy control group.

The delay in the return to baseline of the frontalis muscle is displayed in figure 13 (for means and standard deviations see Appendix D-1).

FIGURE 13



The three groups show a fairly similar pattern for the return to baseline measure, with the stress condition taking the longest time to return to baseline levels. The results of the repeated measures ANOVA (with contrasts as described above) are as follows: The contrast for the two pain groups versus the healthy controls is not significant ($F(1,48) = .066$, $p = .798$), nor is the contrast for the arithmetic versus the other three conditions (stimulus-response specificity assumption) ($F(1,48) = .204$, $p = .653$) or their interaction ($F(1,48) = .329$, $p = .569$). The contrast for the stress conditions versus the neutral condition is, however, significant ($F(1,48) = 5.259$, $p = .023$), but not the interaction ($F(1,48) = .003$, $p = .957$). There are no significant inter- or intragroup comparisons at the preset alpha ($p = .006$) level, albeit a tendency for a significant difference between the alphabet and stress condition in the CBP group to occur ($p = .013$).

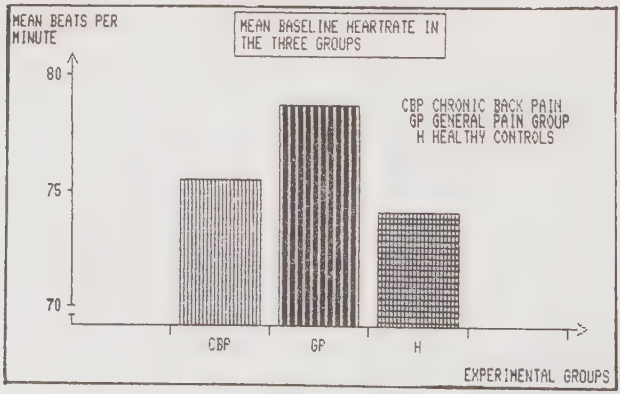
To summarize the data on the frontalis activity for the four experimental conditions and the three groups, only part ot the assumptions of

hypothesis 6 seems to be valid. The frontalis EMG does not show a stimulus-specific response-peak during mental arithemtic, rather, all stress conditions elicit an EMG increase above the level of the neutral condition. This increase is strongest for the stress conditon. The expected higher reactivity of the pain groups versus the healthy controls can not be observed either. The return to baseline values also only yield a significant effect for the conditions, with the stress situation eliciting the longest delay.

6.2.7 Heartrate and Skin Resistance Levels

It was expected that the heart rate responses would be especially strong during the three stress conditions (pain, arithmetic, stress talk) versus the neutral (alphabet) condition, with the pain groups reacting more strongly than the healthy control group. The mean heartrate baseline levels of the three groups do not differ (see figure 14, and appendix D-2).

FIGURE 14



The ANOVA for the baseline heartrate of the three groups yielded a non-significant F-value ($F(2,48) = .545$; $p = .583$).

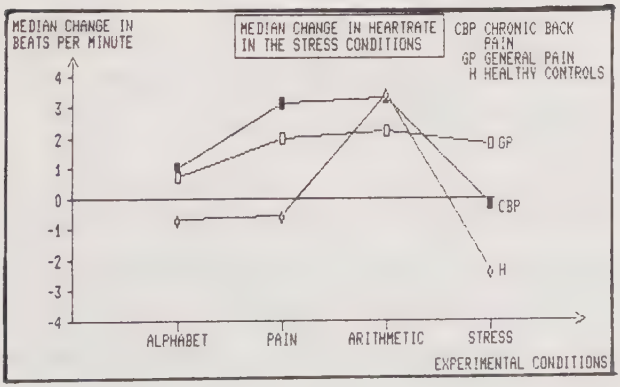
The groups do, however, differ in their reactivity to the stress conditions. Table 27 and figure 15 show the median change in heartrate during stress exposure for the three groups.

TABLE 27

MEDIAN CHANGE IN HEARTRATE (BEATS PER MINUTE) DURING STRESS EXPOSURE

Group	Alphabet Mdn	Pain Mdn	Arithmetic Mdn	Stress Mdn
Chronic Back Pain	1.oo	3.1o	3.3o	-.2o
General Pain	.7o	2.oo	2.2o	1.8o
Healthy Controls	-.7o	-.6o	3.4o	-2.4o

FIGURE 15



The data indicate a similar heartrate reaction for the two pain groups - namely elevation (except for the stress condition in the CBP group) for all four conditions - while the healthy controls do react with heartrate decreases to all but the mental arithemtic condition, which elicted a strong increase. The results of the MANOVA and ANOVA with contrasts (the three stress conditions versus the neutral condition, the pain group versus the healthy controls) are displayed in table 28.

TABLE 28

MANOVA AND CONTRASTS FOR THE CHANGE IN HEARTRATE DURING THE EXPERIMENTAL CONDITIONS

Factor	Wilk's Lambda (1) Mean Square (2)	df	F	p
Groups	117.671 (2)	2	1.35o	.269
within	131.593 (2)	47		
Conditions	.868 (1)	3,45	2.279	.o92
Group x Cond.	.84o (1)	6,9o	1.826	.237
Groups (contrast)	14o9.812	1	2.678	.1o8
within	526.372	47		
Cond.(Contrast)	23.245	1	5.o65	.o29
Group (Contrast) x Cond. (Contrast)	1o.349	1	2.255	.14o
within	4.589	47		

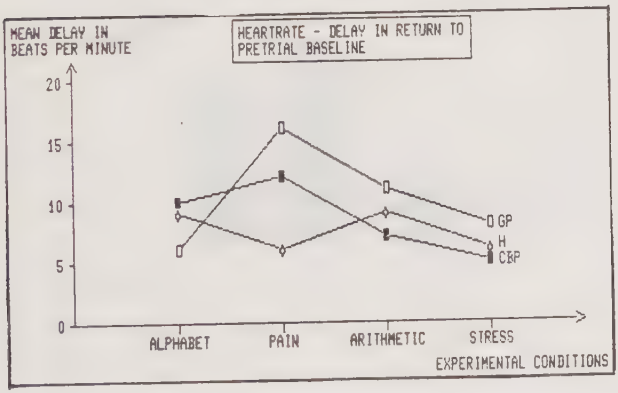
The data show a significant conditions effect and a trend towards significant group and interaction effects when the contrasts are applied.

U-tests do not yield any significant group differences (α set at .oo8). Wilcoxon tests show a significant difference between the stress and mental arithemtic ($p = .oo5$) and a close to significant difference between the alphabet and the mental arithmetic condition ($p = .o1o$) only in the healthy control group. Thus, while the two pain groups respond with undifferentiated heartrate increase (except for the CBP group in the stress condition), the healthy control group responds with heart

rate increase to the mental arithmetic condition only, but with heart-rate decrease to the other three conditions. Thus, the predictions of hypothesis 6 could partially be found.That is, the two pain groups differ from the healthy control group but,contrary to the prediction, they show arousal in almost all conditions - regardless if stress is presented or not - and the healthy controls also react with arousal in the arithmetic condition, but not the others.

The time to return to baseline for the heartrate does not correspond to the heart rate reactivity as figure 16 shows (Appendix D-1 displays the means and standard deviations).

FIGURE 16

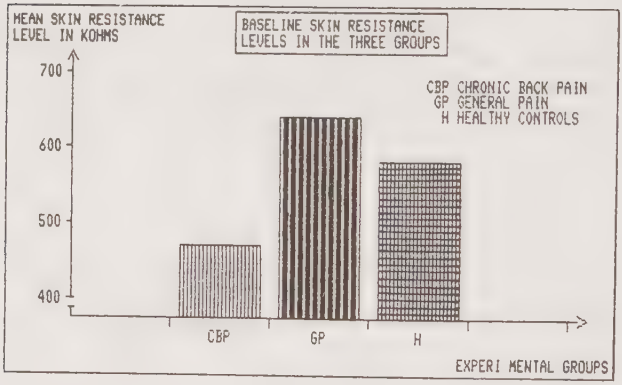


The ANOVA with the same contrasts that were applied to the heartrate change during stress yields the following results: F (1,47) = 1.593, p = .28o for the group main effect; F(1,47) = 1.453, p = .234 for the conditions; and F(1,47) = .866, p = .357 for the interaction. Post hoc comparisons do not yield any significant results except for a tendency towards longer delay of the general pain group, in the pain and arithmetic conditions versus the alphabet (p = .o14 and .o1o, respectively).

Hypothesis 6 had postulated results for the skin resitance levels that are comparable to the heartrate measure: decrease in resistance during all stress reactions with a prominence of these reactions in the pain groups. Figure 17 displays the skin resistance baseline levels

in kiloohms for the three experimental groups. ANOVA reveals that the three groups show a trend towards a significant difference (CBP : M = 469.29, SD = 19o.46; GP : M = 639.19, SD = 235,49; H: M = 58o.67, SD = 221.14; F(2,42) = 2.322, p = .11o5), with a significant difference between the back pain (CBP) and the general pain (GP) groups (p = .o2o) and a trend towards significance between the CBP and healthy control group (p = .o92).

FIGURE 17



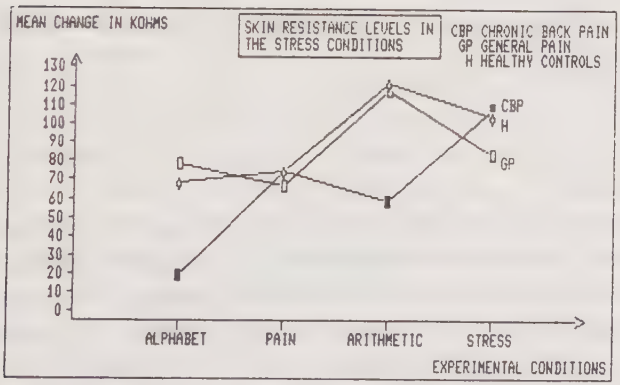
The reactivity of the skin resistance level during stress is displayed in table 29 and in figure 18.

TABLE 29

MEAN CHANGE IN SKIN RESISTANCE LEVELS DURING THE EXPERIMENTAL CONDITIONS IN THE THREE GROUPS (IN KILOOHMS)

	Alphabet M (SD)	Pain M (SD)	Arithmetic M (SD)	Stress M (SD)
Chronic Back Pain	19.3o8(37.o14)	74.692(73.184)	59.ooo(72.869)	1o7.462(9o.219)
General Pain	78.313(13o.473)	65.938(56.693)	117.937(1o4.932)	83.813(1o8.895)
Healthy Controls	68.933(75.1o6)	74.o67(58.316)	127.133(144.132)	1o4.333(1o9.875)

FIGURE 18



The data indicate that the CBP group has a very low reaction to the alphabet condition, a medium high reaction to the pain and arithmetic condition, and a very high response to the stress condition. The CBP group's values tend to be lower than those of the two other two groups which may be due to its initially lower baseline levels. The other two

groups show low reactions to the alphabet and pain and stress conditions. The MANOVA results as well as contrasts for the pain groups versus the healthy group and the neutral versus the stress conditions are displayed in table 3o.

TABLE 30

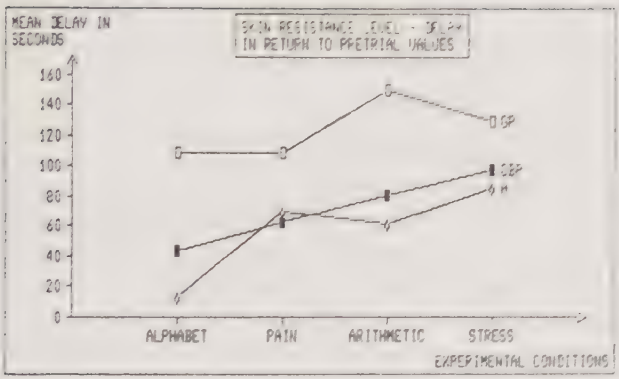
MANOVA AND CONTRASTS FOR THE SKIN RESISTANCE LEVELS DURING STRESS

Factor	Wilk's Lambda (1) Mean Squares (2)	df	F	p
Groups	11281.47o (2)	2	.615	.545
within	1833o.818	41		
Conditions	.748 (1)	3,39	4.377	.oo9
Group x Cond.	.849 (1)	6,78	1.1o7	.366
Group (contrast)	37772.864	1	.515	.477
within	73323.243	41		
Cond. (Contrast)	3453.864	1	5.397	.o25
Group x Cond. (Contrast)	83.716	1	.131	.719
within	639.911	41		

The MANOVA and the contrasts show only a significant conditions main effect, but no significant group or interaction effects. U-tests do not become significant, i.e., there are no group differences. Wilcoxon-tests show that the neutral condition differs very significantly ($p = .oo1$) from the stress condition only in the CBP group, but show no other significant differences.

The delay in the return to baseline for the three groups is displayed in figure 19. Means and standard deviations can be found in Appendix D-1.

FIGURE 19



The (M)ANOVA yields a close to significant group effect ($F(2,43) = 2.842$, $p = .069$) that loses significance with the suggested group contrast (CBP, GP versus H: $F(1,43) = 2.523$, $p = .120$) and a very significant conditions effect ($F(3,40) = 5.321$, $p = .004$) that remains significant with the suggested contrast ($F(1,43) = 5.741$, $p = .021$) which compares the neutral with the stress conditions. Both interactions are insignificant ($F(6,80) = .568$, $p = .754$; $F(1,43) = .697$, $p = .408$). Post hoc single group and condition comparisons show a significant difference between the alphabet and stress condition in the CBP group and the healthy control group, and between the alphabet and the arithmetic condition in the general pain group (preset alpha = .008).

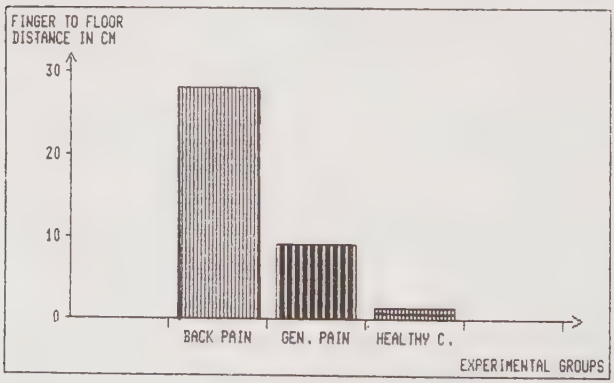
In summary, the results on the heartrate and skin resistance measures are as follows: for both measures a significant conditions effect can be found if the neutral condition is contrasted with the stress conditions. While the heartrate most clearly increases during the mental arithmetic task, skin resistance levels decrease in all conditions, with the reaction becoming stronger from the neutral to the pain, the arithmetic and the stress condition. The group differences approach significance for the heartrate if the two pain groups are contrasted with the healthy control group. The skin resistance level does not yield significant group

differences, although the return to baseline is somewhat prolonged in the general pain group which may, however, be due to this group's very high initial baseline levels. The return of the heartrate to baseline is only a little delayed, with no significant group or condition effect.

6.3. Results on The Behavioral Level

Hypothesis 8 suggests that the CBP patients are more immobile than the other two groups and that this immobility is positively correlated with their anxiety, pain and muscle tension levels. The diathesis-stress model would suggest a relationship between fear of movement or activity-related pain, muscle tension, immobility and pain levels for the CBP patients. Figure 2o displays the results of the immobility measurement - the means for the finger-to-floor distance for the three groups.

FIGURE 2o
FINGER TO FLOOR DISTANCE IN THE THREE GROUPS



While the mean finger-to-floor distance in the CBP group is 27.88 centimeters (SD = 25.39, range 0 to 96 cm), the average distance in the GP group is 9.oo cm (SD = 9.91, range 0 to 33 cm) and that of the healthy controls is 1.35 cm (SD = 3.43, range 0 to 13 cm). The ANOVA is

highly significant ($F(2,48) = 12.607$, $p = .000$) and Tukey tests are significant at the .01 level for the comparison of the CBP with the two control groups.

To assess the relationship of the immobility and the pain ratings, the muscle tension levels, and the anxiety levels of the CBP patients, correlations were computed between these variables. The state anxiety scores were used as an esimate of the activity-related anxiety. The finger-to-floor distance of the low back pain patients correlates positively with the state anxiety score ($r = .588$, $p < .01$), the pain card index ($r = .712$, $p < .001$) and the McGill affective dimension ($r = .572$, $p < .05$) but not with the baseline back muscle tension levels ($r = -.004$ for the left and $r = .085$ for the right back). It is, however, positively correlated with the muscular reactivity of the right back ($r = .353$) and slightly with that of the left back ($r = .146$) although these correlations are not significant with 17 subjects. The finger-to-floor distance is also correlated with the Health Locus of Control powerful other dimension ($r = .552$, $p = .020$) and the negative adjectives of the Depression Adjective Checklist ($r = .505$, $p = .033$).

To assess the relationship of the finger-to-floor distance to organic variables, chi-square analyses or Spearman rank correlation coefficients were computed. The finger-to-floor distance is not significantly related to the presence of back surgery (Fisher's exact test, $p = .664$), or the duration of the pain ($r = .085$, $p = .386$) in the CBP group, but it is significantly related to the number of surgeries ($r = .470$, $p = .029$) and almost significantly to the amount ot degenerative change ($r = .409$, $p = .058$).

In summary, the results for hypothesis eight are as follows: The CBP group displays greatly reduced spinal mobility which is clearly related to the amount of pain and the state anxiety scores, but is uncorrelated with the baseline EMG levels, and is only weakly correlated with the back muscle reactivity. It is also related to an external locus of control and depressed mood, but has a small relationship to the organic variables that were assessed (number of surgeries and degenerative change). The finger to floor distance - just like the paravertebral muscular responsiveness thus seems to be predominantly influenced by psychological variables and less by disease characteristics. A regression analysis, en-

tering organic as well as psychological variables confirms this assumption. The organic variables alone (presence of surgery (number of surgeries, amount of degenerative change and pain duration) only explain 15 % of the variance of the finger-to-floor distance, while the psychological variables (pain card index, McGill affective dimension, state anxiety and state depression) alone explain 71 % of the variance ($F(4,11) = 10.313$ $p = .001$). Inclusion of organic variables does not increase the variance explained considerably (to 78 %).

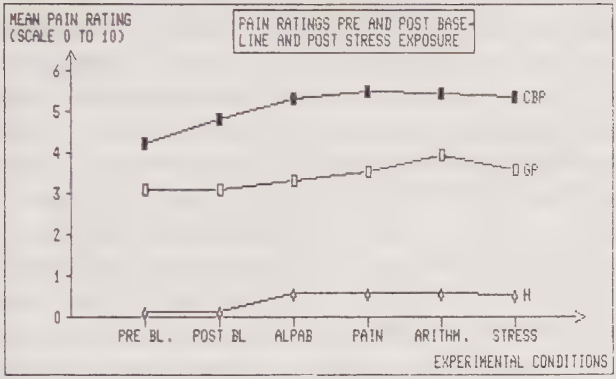
6.4. Results on The Verbal-Subjective Level

6.4.1. Ratings During the Experimental Session

Hypothesis 9 discusses the experimental reactions that were expected on the verbal-subjective level of behavior. Specifically, it was hypothesized that the subjective pain, tension, stress and effort ratings that were taken before and after the baseline and after each experimental condition would be higher in the personally relevant stress conditions (especially tension and unpleasantness), but also (especially the effort rating) in the general stress condition vs. the neutral condition. It was also expected that the pain patients would show higher ratings on these variables than the healthy controls. The pain patients were also expected to be more anxious than the healthy controls.

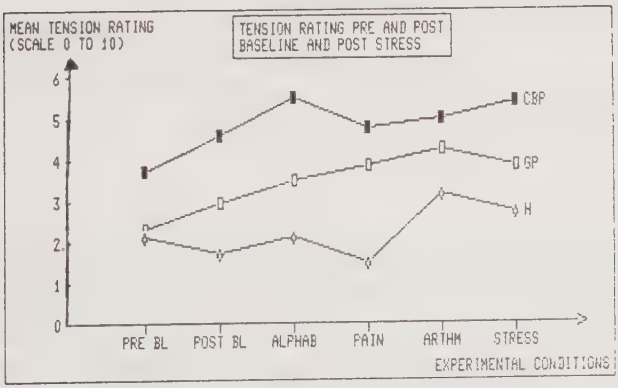
Figures 21 - 24 show the pain, tension, stress ("unpleasantness") and effort ratings of the three groups for the baseline and the four experimental conditions (for means and standard deviations see Appendix E-1)

FIGURE 21



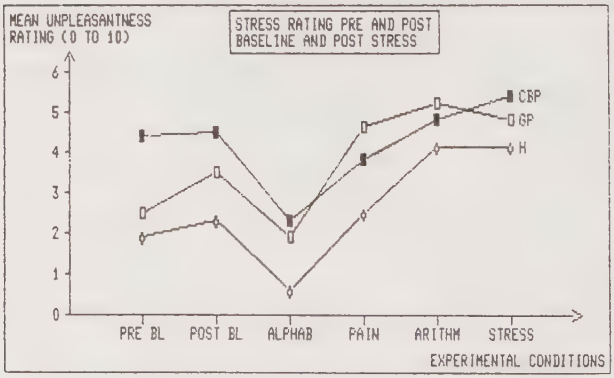
The pain ratings show that the two pain groups experience moderate levels of pain at the beginning of the experimental session with the CBP group displaying not significantly higher levels than the GP group, while the healthy controls experience only minimal pain. The pain ratings increase significantly from the baseline to all experimental conditions only in the CBP group ($p < .05$) without significant differences between conditions. In the GP group there is a significant increase only in the arithmetic condition, while the healthy controls do not respond with pain.

FIGURE 22



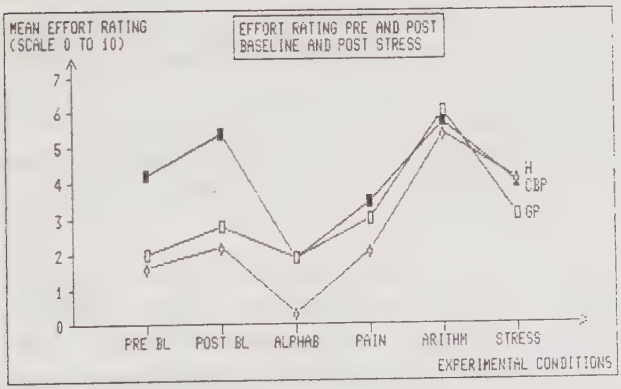
The tension ratings also show a strong increase from the baseline to to the post-baseline and the experimental conditions. The initial tension ratings do not differ for the general pain and the healthy control group, but the CBP group has a significantly higher level (contrast: $F(1,48) = 5.698$, $p = .021$). For the experimental conditions alone a significant group effect (CBP versus GP versus H : $F(1,48) = 10.765$, $p = .002$) but no significant conditions effect can be demonstrated (stress versus nonstress: $F(1,48) = .334$, $p = .566$). The interaction is almost significant ($F(1,48) = 3.923$, $p = .053$). Although the tension ratings increase very strongly from the baseline to the experimental conditions ($p < .05$) in both pain groups, there are no significant between-condition differences. The healthy controls display low tension in the baseline, the neutral and the pain condition (pain is obviously not a potent stressor for them) but significant increases during the mental arithmetic and the stress condition ($p < .05$). The CBP group's ratings are significantly higher than those of the healthy control group in all but the mental arithmetic conditions, and they differ significantly from the general pain group in the alphabet condition.

FIGURE 23



The stress ("unpleasantness") ratings differ between the groups at the pretrial baseline. The CBP group displays a significantly higher level than the two control groups ($F(2,48) = 5.541$, $p = .007$; Tukey tests are significant at the $p = .05$ level). ANOVA for the experimental conditions yields an almost significant group effect (CBP and GP versus H: $F(1,48) = 3.896$, $p = .054$) and a significant conditions effect (contrast for the stress conditions versus the neutral condition: $F(1,48) = 9.086$, $p = .004$). In all three groups the alphabet condition differs significantly from the three stress conditions, implying successful stress induction. This impression is also confirmed by the GSR and heartrate reactions (see 6.2.6). There are no significant between group differences in any condition, except for the alphabet condition where both pain groups have higher scores ($p < .05$).

FIGURE 24



The effort ratings at the preexperimental baseline are again higher for the CBP than the two other groups ($F(2,48) = 4.969$, $p = .010$; Tukey tests are significant below the .05 level). During the experimental conditions all groups show comparable ratings (no significant group effect) but a very significant conditions effect (contrast stress versus non-stress conditions ($F(1,48) = 66.636$, $p = .000$). The mental

arithmetic condition elicits the highest effort rating and differs significantly from all other conditions ($p < .01$) and all three stress conditions differ significantly from the neutral (alphabet) condition ($p < .05$). Again the two pain groups do not differ from the healthy controls in the stress condition, but they do have higher ratings in the neutral condition ($p < .01$).

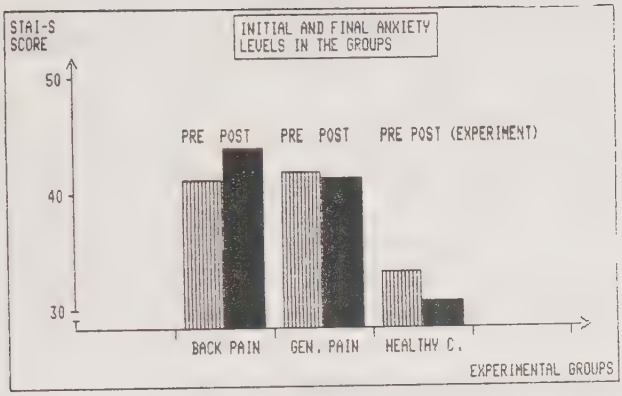
In summary, the results for the subjective ratings before and after the experimental conditions are as follows: At the preexperimental baseline the CBP group shows higher pain, tension, stress and effort ratings than the general pain or healthy control group.

During the experimental conditions the pain levels increase significantly only in the CBP group, the tension levels increase in both pain groups without condition difference. The healthy controls show high tension only in the mental arithmetic and the stress condition.

The stress ratings do differentiate between the neutral and stress conditions in all three groups thus suggesting successful stress induction, with the GP and CBP group displaying higher ratings only in the neutral condition. The effort rating is also very high in the stress conditions as compared to the neutral condition with a peak in the mental arithmetic task. Overall, all the stress tasks elicit much higher stress and effort ratings than the neutral task, but there is no difference between the general or personally relevant stress, nor between the three groups.

Another subjective measure was the State Anxiety Inventory that had been presented to all subjects before and after the experimental session. Figure 25 shows the mean pre and post experimental STAI scores for the three groups (see also Appendix E-2). The ANOVA comparing the groups (using a linear contrast) and the times of presentation yields a significant group effect ($F,48) = 13.557$, $p = .001$), a nonsignificant effect for the time ($F(1,48) = .029$, $p = .865$) and a significant interaction ($F(1,48) = 9.674$, $p = .003$), indicating a differential effect for the groups. The two pain groups display very high initial anxiety levels which increase towards posttreatment only in the CBP group ($t_{(16)} = -3.310$, $p = .004$) while the anxiety does not change significantly in the two other groups, though there is a trend towards a decrease in the healthy control group ($t_{(16)} = 1.78$, $p = .094$).

FIGURE 25



Additionally, the stress situations that were presented by the subjects were classified according to the principal emotion displayed in them to determine if any specific conflicts and emotions predomianted among the stress stimuli and reactions. Twenty-five times anger (about the family, collegues or doctors) was the predominant emotion, 16 times anxiety (about the well-being of the family, one's own or a family member's health) was discussed, depression and helplessness (about familial, job- or health-realted problems) were the topic seven times and embarassment (in a sexual, a job-related or a medical situation) was the topic three times. The distribution of the emotions in the groups can be found in table 31.

TABLE 31

DISTRIBUTION OF THE PREDOMINANT EMOTIONS IN THE STRESS SITUATION IN THE THREE GROUPS

	Anger	Anxiety	Depression Helplessness	Shame
Chronic Back Pain	5	7	4	1
General Pain	1o	5	2	o
Healthy Controls	1o	4	1	2

Anger is the predominant emotion in the general pain and the healthy control group while anxiety is most dominant in the CBP group, followed by anger, helplessness and depression. There are no significant group differences in the predominant emotion.

Interestingly, eight patients in the CBP group report anxiety and worry about their own health (N = 3) or the health of a family member (N = 5) as compared to one each in the healthy and general pain group worrying about the health of a family member. Three patients in the GP group but none in the healthy control group report to be anxious about their own health. This difference in preoccupation with health-related concerns is significant at the .o5 level between the groups.

6.4.2. Appraisal, Coping and Muscular Reactivity

Hypothesis 1o had suggested that the amount of back muscle reactivity would depend on the ability to cope with stressful and painful stimulation in the CBP patients. To test this hypothesis, various psychological variables that were thought to be indicators of the ability to cope with and the appraisal of painful and stressful stimulation - such as the health locus of control (HLC), the Pain Experience Questionnnaire Score (as an indicator of the cognitive and affective evaluation of the pain experience), the McGill affective and evaluative components (as indicators of the ability to cope with pain), state anxiety (STAI) and depression (BDI or DACLT) were entered into a multiple regression analysis.

The mental status rating as well as the cognitive and physical coping skills ratings from the Pain Assessment Report could not be used due to too many missing values. The dependent variable was the combined reactivity of the right and left back musculature to the personally relevant stress conditions. Table 32 displays the results of this procedure.

TABLE 32

RESULTS OF THE REGRESSION ANALYSIS FOR THE BACK MUSCLE REACTIVITY IN THE CBP PATIENTS

Variables	ß	R	R^2	adjusted R^2	df	F	p
DACL Total Score	.865	.945	.894	.745	6,4	5.65o	.o5
HLC Powerful Other	.555						
Pain Exoerience Questionnaire	.216						
McCill Evaluative Component	-1.292						
State Anxiety	-.562						
McGill Affective Dimension	1.178						

The hypothesized psychological variables explain 73 % of the variance of the muscular reactivity, whereby BDI or DACL are exchangeable - with DACL yielding a slightly higher coefficient.

A subsequent stepwise regression analysis that was used to determine the most relevant variables showed that the depression (BDI) alone explains 48 % of the variance (adjusted for small N) ($R = .744$, $R^2 = .554$ adjusted $R^2 = .479$, $F(1,7) = 14.833$, $p = .oo6$; $ß = .825$) and the combined BDI and PEQ (Pain Experience Questionnaire) scores explain 76 % of the variance ($R = .9o9$, $R^2 = .826$, adjusted $R^2 = .756$, $F(2,5) = 11.861$, $p = .o13$) ß (BDI) = 1.oo9, ß(PEQ) = .582). This means, that patients with high muscular reactivity are those who are most depressed and who are most worried and emotionally affected by their pain problem and probably also other forms of stress. Of course, the direction of this relationship is not clarified.

To assess the relationship between back muscle reactivity and psychological components still further (especially for the remaining subjects who fell out of the regression procedure due to missing values), the CBP patients were divided into clearly identifyable "responders" and "nonresponders" according to the reliability of their assignment into the CBP versus the other groups on grounds of their muscular reactivity. These t-tests between responders and non-responders yield significant differences in almost only the psychological variables suggested above. The

responders score higher than the nonresponders in their state anxiety scores (M = 45.5o (SD = 9.89) versus M = 36.8o (SD = 5.72), t(13) = 2.27, p = .o21), the Depression Adjective Checklist total score (13.25 (5.328) vs. 1o.2o (1.3o) t(14) = 1.85 p = .o4o), and the negative adjectives score (5.5o(4.34) vs. 1.oo (.71) t (12) = 3.48, p = .oo3) and the Health Locus of Control powerful other dimension (21.17 (8.21) vs. 12.8o (7.69) t(14) = 1.85, p = .o4o) with a trend towards a significant difference in the McGill evaluative dimension (.65 (.32) vs. .5o (.11), t(14) = .1o) and the differences in the other suggested variables (e.g., PEQ, Mc Gill affective dimension, PEQ) pointing in the predicted direction.

To assess the relevance of organic variables for the responders versus nonresponders, the variables presence of surgery, number of surgeries, amount of degenerative change and duration of the pain were entered into a multiple regression procedure with a subsequent backward exclusion procedure. No significant regression function could be found by either method. Organic variables also did not differentiate the responders from the nonresponders when t-tests were used.

To summarize the data on hypothesis 1o, the following results should be noted: the CBP patients' muscular reactivity of the back is highly related to their mood, the cognitive appraisal of and the affective reaction to their pain experience, their locus of control and their anxiety level. These variables were considered to be indicators of their ability to cope with aversive stimulation. Given this assumption is justified, a strong relationship between the patients' coping ability and their spinal muscular reactions to stress can be noted. Organic variables on the other hand could not predict the muscular responsivity nor could back muscle "responders" and "nonresponders" be differentiated by them.

However, it could not be clarified why about 23 % of the CBP patients do not show abnormal back muscle activity, but nevertheless have high pain ratings. Close analysis of the four clear nonresponders reveals that one of them had very extreme EMG levels (28.8 microvolts left back baseline) which may have led to a ceiling effect. Another patient could not reproduce any stressful situations (stress ratings of o-2 only) which may explain the low reaction. For the other two patients no deviance from the group scores could be noted. Their pain may be under control of variables not assessed here. They may be organic - or perhaps operant

factors that could not be evaluated here due to too many missing values in the "operant behavior" scale.

7. Discussion

7.1. Summary and Interpretation of the Results

The purpose of the present study was to investigate the psychophysiological responses in chronic back pain (CBP) patients within a biopsychosocial framework. Specifically, a preliminary investigation of the adequacy of a diathesis-stress model of CBP was undertaken. It was hypothesized that CBP patients would display abnormal reactions of their back musculature (strong EMG increase, long delay in return to baseline) when exposed to personally relevant stressors as compared to healthy controls and other pain patients (without back involvement). It was further hypothesized that they would not differ from other pain patients in non-back-related physiological variables (e.g., frontalis EMG, heartrate, skin resistance level). In addition, it was expected that the high muscular reactivity would be related to poor coping ability and that the CBP patients would show high spinal immobility that was thought to be related to high anxiety, pain and muscle tension levels. Furthermore, higher baseline EMG levels were expected for the CBP group though they were not considered as central characteristics of this population as were the reactivity and return to baseline. No differences in baseline tension or muscular responding were expected between "organic" and "nonorganic" CBP patients.

The results of this study fully confirm the predictions of the first two hypotheses, namely, that CBP patients show abnormal back muscle reactivity to personally relevant stress and that they show prolonged delay in return to baseline in their back EMG values. Both, change in EMG level and time to return to baseline during the pain and stress discussion, are clearly elevated only in the CBP group while the non-back pain group and the healthy controls do not show any abnormal back muscle activity at all. This suggests that the abnormal paravertebral muscle response is typical for the CBP patients. It is obviously not a phenomenon of chronic pain in general (since the general pain (GP) group shows hardly any reaction), nor is it part of a general stress reaction (the healthy controls indicated comparable levels of stress but no back muscle response). This specific paraspinal muscular reaction of the CBP patients can also be demonstrated by the fact that the majority of the CBP patients can be reliably classified into the CBP group versus the general pain and healthy control groups, on grounds of their back muscle activity alone.

This abnormal activity also seems to be specific for the back only. Although the CBP group shows a pattern in the frontalis musculature which is similar to that of the back, the frontalis reaction is much less pronounced and not significantly different from that of the other groups. Rather, the frontalis muscle shows a strong response in all stressful conditions, especially in the personal stress condition. Thus the assumption of a specific paravertebral stress reaction in CBP patients seems to be warranted. Still better evidence for this assumption would come from multi-site EMG recordings that could not be performed in this study.

It was also hypothesized that this abnormal back muscle reaction would only occur in stress situations that were of special emotional relevance to the patient as in the pain and personal stress condition. It was not expected to be elicited by the mental arithemtic task which requires some degree of mental effort but little emotional involvement. The negative results of the Collins et al. (1982) study may be attributed to this aspect. They did not find abnormal back muscle activity in CBP patients when they were exposed to a mental arithemtic task or the cold pressor test. This study confirms their result in that on the average the CBP group does not show an abnormal back muscle reaction to the mental arithmetic task, although a few patients did react especially when they were embarrassed about their inability to complete the task. Overall, the back muscle reaction to the mental arithmetic task does not differ significantly from that of the neutral task, although this task did elicit high "unpleasantness" and effort ratings as well as high GSR and heartrate responses. Contrary to the Collins et al. (1982) study the CBP patients do show strong paravertebral muscle activity during the pain task. However, talking about a recent pain episode differs considerably from the cold pressor test used in the Collins et al. (1982) study. The cold pressor task uses experimental pain that is unlikely to evoke the same emotional reactions as clinical pain. Besides, the extremely short exposure time of 3 x 12 seconds may not have been sufficient for a pain experience to develop at all. They also failed to assess any stress or pain ratings. Due to this difference in experimental stimuli the results of this study do not necessarily contradict those of the Collins et al. (1982) study.

Although this study found strong reactions of the back musculature

during discussion of painful and stressful experiences, it could not clarify why the CBP patients reacted very strongly with their back muscles only to the self-reported stress as compared to the experimenter-induced stress. It is, however, assumed that only the self-reported pain and stress episodes were of personal relevance to them. This assumption may follow from the cognitive-behavioral perspective assumed in this study. The experience of stress is believed to depend not only on aversive stimulation but also the person's appraisal of the stimulation and the availability of coping strategies. It is conceivable that the laboratory situations were not equally stressful for all patients and that they did not elicit as strong and specific emotional reactions as did the self-reported stressful events. Almost all pain and stress situations reported in the group elicited a strong emotional reaction with anxiety and anger being the predominant emotions. This supports the observations of Janus (1975 , 1978) and Dorpat and Holmes (1962) who presented, however, only anecdotal evidence. The contents of the stress-situations were dominated by concerns about the health of a family member or the patient and problems in the family or the work place.

Interestingly, the variance of the back muscle reaction is very high in the CBP group. This is mainly due to the circumstance that about 77 % (N = 14) of the CBP patients show very abnormal reactions of their back musculature, while about 23% (N=3) react similarly to the general pain group or the healthy controls. Hypothesis 1o had suggested that differences in appraisal of stressful (including painful) stimulation in coping with stress may be responsible for the differences in reactivity. Furthermore, patients with high anxiety and depression scores, high external locus of control and with very negative emotional and cognitive evaluations of their pain could be considered as bad copers who were expected to show higher muscular reactions. This was supported by the results. The amount of back muscle reaction to stress was found to be significantly related to high anxiety, high depression, external locus of control and negative evaluation of the pain experience(McGill evaluative and/or affective dimension and/or Pain Experience Questionnaire Score). It was not related to the organic variables that were assessed in this study. Still, no satisfactory explanation could be found why 23 % of the CBP patients do not show an abnormal stress reaction of their

back musculature. While one patient's EMG levels may have reached the ceiling (baseline values of up to 29 microvolts) and another patient was not aroused by remembering previous painful or stressful situations, no explanation could be found for the lack of reactivity of the other nonresponders. Although the non-responders possess better stress coping abilities, they still experience levels of back pain that are comparable to those of the responders. However, they do show less affective pain responses. The organic variables that were assessed in this study are not more prominent in this group, though not all possible organic variables may have been measured. Another possibility is that their pain may be under operant control . This assumption could not be investigated in this study, due to too many missing values in an operant behavior scale that had been given to some patients and to a small number of nonresponders. It cannot be expected that in as heterogeneous a back pain group as the one assessed in this study all patients will exhibit a uniform psychophysiological pattern. It remains the task of further research to determine subgroups of back pain patients with specific psychophysiological characteristics. An additional complication of this study is that all patients were assessed after they had developed chronic back pain. It can therefore not be determined whether the abnormal muscular reactivity is the cause or the consequence of their back pain. Prospective studies are needed to determine this relationship.

Hypotheses 3 and 4 had postulated that CBP patients may show elevated resting tension levels in their back musculature that may be lowered with increasing pain duration. Additionally, it was suggested that patients with an organic diagnosis would not differ from those without it. The data indicate that the CBP patients' paravertebral muscle tension levels are partially higher than those of the control subjects, i.e. , as far as the left back or the affected side(s) are concerned. However, the levels are generally rather low (in the 2 - 4 microvolt range). They are correlated negatively with duration of the pain although the correlation does not reach significance. It is possible that muscle fibers degenerate with time and lead to lower EMGs, as was predicted by Fassbender (1973, 1975). This study could neither confirm nor refute this possibility. It could also make no statement about the question whether muscle spasms in CBP patients can truly be evaluated by surface electrodes. Hypotheses 8 had suggested that CBP patients have low spinal mobility that may be correlated with their anxiety of movement, their pain level, and their back muscle tension

levels. Most of the predictions of this hypothesis could be found. The CBP patients are more immobile as compared to the other subjects, and the amount of immobility of the low back pain patients is positively correlated with their anxiety and pain levels, but not with the baseline muscle tension levels. There is a positive - though not significant - relationship to the muscular reactivity of the back. The predictions of hypothesis 8 could thus be supported to a large extent.

The frontalis, heartrate and GSR measure as well as the subjective ratings show that the induction of stress was successful for all stress conditions. All ANOVAS yielded a significant condition effect for these physiological and subjective measuresof arousal. An interesting effect could be found in the heartrate: while both pain groups reacted with heartrate increase to all stress conditions (except for a minimal heartrate decrease in the CBP group in the stress condition) the healthy controls reacted with decrease to all but the mental arithmetic condition, which elicited a strong response. In the GSR values an increase in all conditions, but expecially the stress conditions could be observed. The heartrate seems to respond mainly to the effort required for the task, which seems to be much higher in the pain groups than in the healthy controls - except for the mental arithmetic, which was experienced as equally straining. In contrast, the GSR much rather seems to tap the general arousal elicited by a task. Contrary to the findings of Collins et al. (1982), we could not find a stronger frontalis or skin resistance reaction in the CBP group as compared to the other groups. However, the stressors used in this study did differ from those of the Collins et al. (1982) study, in exposure time as well as method. We also used MANOVA, or Geisser-Greenhouse-corrected ANOVA, and change scores as compared to Collins et al. (1982) who used uncorrected ANOVA (thus possibly overestimating effects if the variances were heterogeneous; this cannot be assessed since they do not report means and standard deviations) and absolute values. Contrary to earlier findings (cf., Passchier & Helm-Hylkema, 1981), we also could not find a strong frontalis response for the mental arithemtic task. However, the subjects in this study were not prompted and had their eyes open during the task, which may have changed their frontalis activity. Overall, the three groups show a trend toward a differential reaction in the heartrate, with the pain patients displaying a tendency to uniform heartrate increase while the healthy controls show decrease except for the mental arithmetic task. The sub-

jective stress and effort ratings do not differ significantly between the groups. Thus the differential back musculature reaction cannot be attributed to differences in severity of the reported stressful situations.

Overall, most predictions related to the back muscle reactivity in CBP patients were found to be true. These results are compatible with most previous research. The conflicting findings of previous studies be due to the divergent stress stimuli used. Never before have self-selected stressors been employed in a laboratory psychophysiological assessment procedure with chronic back pain patients . While Janus (1975, 1978) or Dorpat and Holmes (1962) had probably elicited personally relevant stress situations during psychoanalytic interviews and had found abnormal muscular reactivity, Grabel (1973)and Collins et al. (1982) used threat of shock, mental arithmetic or the cold pressor test as stress stimuli without assessing their emotional relevance for the subjects, and had not found any specific back muscle response. This study found abnormal back muscle reactivity in self-generated reports on previous painful or stressful episodes, but not during an experimenter-imposed mental arithmetic task. Thus, only specific stressors may elicit an abnormal back muscle response. As previously suggested (e.g., Philips, 1977), differences between the groups could much more clearly be found in the reactivity to stress and the time to return to baseline than in baseline resting tension levels. The findings of this study suggest that abnormal reactions to stress and delayed return to baseline of the paraspinal muscle tension levels may be much more instrumental in the development or maintenance of CBP than elevated resting tension levels. The following section will discuss the implications of these findings for the diathesis-stress model of CBP, then methodological limitations of the study will be discussed, and finally implications for diagnosis, therapy, and research on CBP will be pointed out.

7.2. The Relationship of the Results of the Study to the Diathesis-Stress Model of CBP

The diathesis-stress model of CBP , as described in section 4.2. (see page 25), had suggested that the pathogenesis of CBP could be explained in the following way: prolonged or recurrent potentially aversive stimulation may lead to a back muscle response stereotypy, provided that the

stimulation is perceived as stressful and that a diathesis of the back exists (be it in the form of previous trauma, learning or a congenital vulnerability). Repeated muscular hyperreactions to stress are believed to lead to long-lasting ischemia and the release of pain-eliciting substances. The pain reaction may lead to more stress and tension and in the long run also to fear of movement and acitivity, to immobility and thus more muscle tension and a vicious stress-tension-pain-inactivity cycle may ensue.

In this study the experience of aversive stimulation was re-created (by having the patients discuss previous painful or stressful events) - potentially aversive stimulation (mental arithemtic) was presented. The CBP patients did display an increase in back muscle tension and a delay in return to baseline levels in response to talking about previous painful and stressful stimulation (personally relevant stressors) but not in response to reciting the alphabet (neutral condition) or to performing a mental arithmetic task (general stress control). Obviously only specific stressful - probably strongly emotional - stimulation leads to an increase in paravertebral muscle tension over extended periods of time. The specific cognitive appraisal and emotional reaction to the stressor seem to be important for the muscular reaction. These findings give evidence to links a-c of the model. The specific overreaction of the back muscles did not lead to immediate pain-increase in the affected area. Thus link e could not be directly observed. However, the time lag for pain to develop may be much longer than the time of observation. In addition, the stress exposure time was very short. Also, recall of a stressful event is unlikely to elicit as strong a response as the original stressor. Interestingly, the report of a previous pain episode elicited as strong a reaction as the recall of the stressful event. This gives some evidence to links h and e of the model. Furthermore, it was found that pain and spinal immobility are strongly related in the CBP group (see link f) and that the immobility is also positively correlated with the patient's anxiety (links j and k), depression scores, external locus of control (link p) and a tendency to higher muscular reactivity (link i).

Additionally, organic variables exert a minimal influence (e.g., number of surgeries and amount of degenerative change).

A strong relationship was also found between the extent of muscular overreaction of the back , the ability to cope with stress (pain) and the appraisal of stressful (painful) stimulation. Seventy-four percent of the variance in muscular reactivity could be explained by psychological variables ,thus providing evidence for links a-c, e, g and h of the model.

Although, in general, most aspects of the model can be supported by the experimental results several caveats are necessary. Most data are correlational in nature and the assumed direction of the relationship of the variables may be different from the one proposed. It is especially important to note that all patients were assessed after they had developed CBP. It is well possible that the observed reactions are only a consequence rather than an antecedent and a consequence of the chronic pain problem. Prospective studies with acute back pain patients or healthy controls with a tendency for abnormal back muscle reactivity are necessary to answer the question of the pathogenetic relevance of the model. More research is also needed on the organic variables involved in the problem of chronic back pain. Nor could operant factors be assessed as was deemed necessary. Whether development of ischemia occurs prior to or following muscular overreaction has not been examined. Measurements of temperature or blood flow at the affected site may be useful but could not yet be incorporated in this study. The very long times to return to baseline in the back muscles of many CBP patients (often more than 5 minutes after 1 minute of stress exposure) points in the direction of the hypothesized relationship between high muscular reactivity, prolonged tension and subsequent ischemia and pain. Clearly more research is needed to assess these variables in more detail. This research should include examination of the biological substrate of these processes and the integration of these findings with current psychophysiological and psychological theorizing.

Overall, the results of this study are promising. None of the predictions of the model has been falsified by the results except for the lack of relationship between the immobility measure and the resting tension levels of the back muscles. This may, however, be due to EMG measurement problems. On the other hand, although most of the assumed relationships could be found in this study,they are often correlational in nature,and measurement problems are obvious in some

variables. The central prediction of the model - abnormal back muscle reactivity to stress in the CBP patients - was observed. This study is a first attempt to clarify the relationship of biological, psychological and social variables in CBP within the framework of the proposed diathesis-stress model. At this point in time it raises more questions than it can answer. Subsequent research will have to evaluate the suggestions described in this paper more carefully.

7.3. Limitations of the Study

Besides the problems already mentioned above, the study has several limitations that endanger its internal and external validity.

First, the physiological assessment posed several problems. The room temperature was not kept constant nor was it consistently measured, thus precluding its use as a covariate. Therefore the absolute EMG values, and especially the very temperature-sensitive skin resistance values must be viewed with caution. They may have been distorted by varying temperatures. It is, however, unlikely that the temperature changes differentially affected the the muscular reactivity in the groups and across the experimental conditions . All influences should have been randomly distributed in the groups and conditions since the subjects in the three groups were tested at random intervals and the conditions were counterbalanced.

As a further complication, talking proved to be a problematic task. In some instances, it led to strong involvement of the facial musculature, thus influencing the frontalis readings. Also, the talking was sometimes accompanied by hand or body movements although the patients had been advised not to move while talking. Three measures were taken to alleviate the problem as far as possible: (a) recitation of the alphabet was used as a neutral talking control condition and the stress conditions were compared to it; (b) the patients were repeatedly told to speak without moving; (c) if gross movements occured nevertheless, they were marked on the session protocol together with the time period they had occurred in. Before the data analysis was performed, unusual peaks due to movement, coughing or sneezing were removed from the data and replaced by the means of the previous and subsequent 2-second trial.

An additional problem was posed by possible carry-over effects between t experimental conditions due to the repeated measures design. To avoid

this effect each condition appeared equally often in the first, second, third or fourth position of each sequence. Also, each condition was followed by a 5-minute posttrial baseline .Then an additional 2-7 minute period was interspersed. This additional interval was used for new instructions, checks of the equipment, the protocol of the subjective ratings ,and it allowed the patients to change their position and to recover from the strain of the experimental condition. No obvious condition to pretrial baseline carry-over effects could be found when the pretrial baselines were inspected (see Appendix D-1 for means and standard deviations). To examine this impression statistically, an ANOVA with contrasts that simulated carry-over effects between conditions and pretrial baselines was computed (cf., Nie et al. , 1981). The ANOVA yielded a nonsignificant result ($F(1,48)=1.042$, $p= .312$) , suggesting the absence of carry-over effects from one condition to the following pretrial baseline. T-tests fully confirmed this result. There were no significant differences between the pretrial baselines of the four conditions. The same is true for condition-to-condition carry-over. An ANOVA with contrasts simulating increasing carry-over from the first to the last condition yielded a nonsignificant result ($F(1,48)= 1.182$, $p=.282$).

Another problem was the presence of back surgery in several CBP patients. Back surgery is known to lead to denervation potentials in the muscles of some patients (cf., Wolf et al., 1982), resulting in unstable and very variable and unreliable EMG values. The best way to handle this problem would have been to determine the presence of denervation potentials in a neurological investigation, or to perform at least multiple assessments of the paraspinal EMG. None of these procedures could be used. In order to attack this problem with the means that were available ,the initial baseline and the pretrial baseline were inspected. If they differed by more than 500% of the initial value (a difference considerably larger than any one found in the healthy control group), this patient's values were excluded from further analysis. Although this procedure can not guarantee that all patients with denervation potentials were excluded from the study it is rather unlikely that they greatly endanger the validity of the stress-related findings. It may be expected that these potentials cause random variation of the signal. This random variation would only contribute to the overall variance and thus at best bias the results in favor of the null hypothesis.

The unknown reliability and validity of the Pain Experience Questionnaire and the Pain Assessment Report pose additional problems. Findings based on these measures must be viewed with caution as long as the psychometric properties of the scales are not yet established. Also, several instruments were only given to a small number of the patients thus reducing the number of subjects for the statistical analysis to critically low values. An additional problem was that no validated measure assessing the appraisal of and the ability to cope with stressful or painful stimulation was used in this study. All measures that were thought to be indicative of the patient's mode of appraisal and coping actually only indirectly assess these variables.The conclusions based on these instruments must be viewed with caution. The same is true for the State Anxiety Inventory as an indicator of movement- and activity-related fear. The patients' fear of movement was not directly measured and it was only hypothesized - but not proven - that the STAI may pick up this fear. Also, a more comprehensive inactivity and immobility measurement could have been conducted (cf., Moll & Wright, 1980). The question of the adequacy of surface EMG measurement for the assessment of painful muscle spasms has already been addressed in section 4.3.The problematic validity of this measure for the registration of resting(as opposed to phasic) muscle activity poses problems for the interpretation of all results related to the resting muscle tension levels.

The small N (17 per group) was at times problematic. Although it was sufficient for the analysis of change in the experimental conditions, the N became critically low when subgroups of patients were examined. Also, the CBP as well as the general pain group were rather heterogeneous in that they assembled patients with widely divergent diagnoses. Replication of this study with larger numbers of subjects and more homogeneous groups is warranted.

With regard to the relevance of the study for chronic back pain patients in general several problems must be adressed (see also 7.4.). It is important to note that the resting muscle tension levels of the CBP patients were generally very low. The increase of the back EMG during stress exposure was also not very dramatic (in the 2 micro-volt-range). This raises the question whether so small a change - however statistically significant - may have any clinical relevance. It is

not possible to answer this crucial question. The anecdotal evidence from the pre-and posttreatment data of three patients suggests that attenuated muscular reactions to stress stimuli may be instrumental in the reduction of the CBP patients' pain. It would be interesting to examine the back muscle tension levels of unimproved patients in comparison to those of successful participants of a pain management program. Only then could the question about the relevance of the back muscle reactions for CBP be further evaluated. Aside from that it may be hypothesized that the EMG increase will be much more dramatic and longlasting in real-life situations. Further research approaching real-life conditions as closely as possible and longer periods of measurement will help to answer this question.

As mentioned before, the patients went through the experiment only after they had become chronic back pain patients. It is not known whether the abnormal back muscle reactivity precedes or follows the development of CBP. Data on healthy controls with occasional but not yet severe back problems (some of the subjects that had been excluded from the healthy control group) suggest that a tendency to abnormal reactivity may be present before the development of CBP.

An advantage of the study - as far as the external relevance of the results is concerned - is that it included the majority of the representa population of CBP patients. Only patients with inflammatory problems or those with acute neurological involvement were excluded. The results of this study can therefore be generalized to the majority of the CBP patients. Further research will, however, have to determine subgroups of CBP patients with differences in psychophysiological responding, as the nonresponders found in this study suggest.

7.4. Implications for Assessment and Treatment of CBP

Provided that the results of this study are replicable and prove to be valid, they may have important consequences for the assessment and treatment of chronic back pain.

Neither medical nor psychological approaches to the assessment of CBP have so far incorporated a stress-reaction paradigm. Medical diagnoses usually focus on structural or movement-related abnormalities of the spine and the back musculature and will use radiological or neurol-

olgical analyses, among others, to assess the causes for CBP. Psychological assessment focusses on operant variables, the patients' mood, cognitions, coping skills, resting muscle tension levels, and so forth, depending on the type of theoretical approach used. The results of this study suggest that it may be very informative to assess the patient's muscular reactions to personally relevant stress situations, and to relate these reactions to other medical and psychological variables. This method may become the basis of differential indications of various psychological or medical treatments or their combination for subgroups of CBP patients. However, before this procedure can be used as a routine assessment paradigm more research with improved methodology (e.g., multi-site, multi-stressor recordings) needs to be undertaken. This paradigm may also serve as an outcome measure to assess treatment-specific changes in muscular reactivity.

The modification of stress-related muscular hyperactivity of the back may become an important aspect of treatment which has so far not sufficiently been considered. Stress-management including relaxation training, coping skills training, the modification of maladaptive cognitions, and so forth, may be the treatment of choice, given the results of this study. The initial anecdotal evidence presented before supports the notion that this kind of treatment may lead to improvement in the pain problem and concomitant reduction in muscular reactivity. It would also be interesting to assess the contribution of somatic treatments to the alleviation of the muscular hyperactivity and the relevance of the reduction in reactivity for the treatment outcome.

7.5. Conclusion and Suggestions for Further Research

This study contributes to the current literature on chronic back pain in that it proposed a diathesis-stress model of chronic back pain and demonstrated that the results of the experimental investigation based on the model are consistent with it. CBP patients have been shown to display abnormal activity of their paraspinal muscles when they are exposed to personally relevant stress.

This study included several methodological improvements over previous studies. First, it employed a non-back pain as well as a healthy control group to differentiate back-pain-related effects

from those attributable to chronic pain in general. Second, it included unilateral EMG-recordings from several sites and the registration of heartrate and skin resistance levels to allow conclusions to be drawn about the generality as opposed to the specificity of the back muscle response. Third, and most importantly, this study used stress-stimuli that were self-selected by the patients in contrast to experimenter-selected stimulation, and it assessed the subjective distress caused by the stimuli. The study also examined the relationship of organic and psychological factors to the muscular stress response. Furthermore, sample characteristics are described in more detail than in previous studies.

The study replicated findings of previous research, namely the lack of a back muscle reaction to mental arithmetic and the equivocal findings on baseline resting tension levels. It could not replicate the findings of higher frontalis muscle tension and GSR levels from previous studies (cf., Collins et al., 1982). This may - besides differences in the experimental setting and the samples - be due to the consideration of heterogeneous variances in the statistical analyses. MANOVA was used to avoid unjustified bias of the data towards rejection of the null hypothesis.

Throughout this paper suggestions for further research were made. The following list summarizes the most important of these:

1. Replication of the findings of this study is warranted. This should be done with multi-site EMG recordings, the inclusion of temperature and pletysmographic recordings, more homogeneous groups of patients, additional measurements at the pain-relevant site of the non-back pain patients, stress stimuli that are even closer to everyday stress situations, and measurements that are taken in various body positions.
2. The relationship of the muscular reaction to various psychological and organic variables and to the pain experience should be assessed in a methodologically improved way. Specifically, the role of cognitive appraisal and coping processes, as well as that of operant variables, should be examined in an experimental design.
3. Longitudinal research should follow up healthy patients with and and without a tendency to dysfunctional back muscle activity in order to assess the relationship between stress-related back muscle activity and the development of chronic back pain. A

related strategy would be to assess acute back patients according to the paradigm described here and to follow them up for an extended period of time in order to evaluate which variables are instrumental in the development of a chronic back problem as opposed to remission of the problem. The relevance of the stress-related overactivity of the back and various psychological as well as organic variables should be assessed.

4. Assessment of CBP should include a procedure testing the stress-related back muscle reaction before and after various treatments in order to assess its relevance for treatment outcome. The same procedure could be used to define subgroups of back pain patients with specific psychobiological characteristics (among other assessment procedures).
5. Various treatment approaches should be examined in the light of the findings of this study. Stress-management ought to be compared to more conventional treatments directed at the reduction of resting muscle tension levels, for example physical therapy, treatment with muscle relaxants or straightforward EMG biofeedback directed at the reduction of resting tension levels. It would also be interesting to assess the efficacy of biofeedback directly addressing the attenuation of muscular stress-reactions induced during treatment.
6. Last, an integration of research on the psychophysiological level with the current medical research as well as the various lines of psychological research is needed. A truly biopsychosocial perspective will require the integration of many avenues of research in an interdisciplinary effort.

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APPENDIX

APPENDIX

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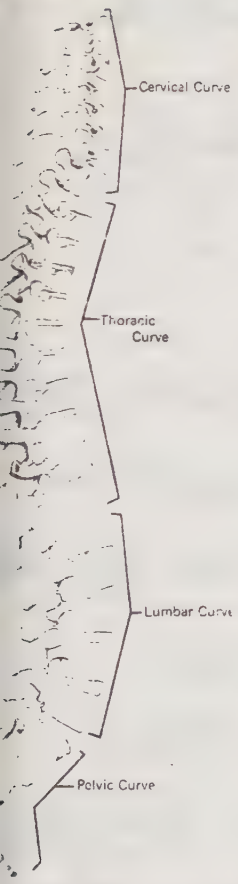
Do you want to know more about your ← back?



Lateral view of the vertebral column.

The Pain Management Program is looking for volunteers with and without chronic pain for a study on the psychophysiology of back pain. Spare 90 minutes of your time and help us help chronic pain patients. If you are interested, please call Roberta Stephens at extension 691. Thank you.

Are you

male, between 30 and 70, and have never had back pain?

Then we need you as a volunteer to participate in a 90 minut psychophysiological assessment in the Pain Management Progra

If you are interested, call Roberta Stephens at x 691. Thank you!

PAIN BEHAVIOR CHECKLIST

DATE_____

CLINICIAN_____ PATIENT_____

Pain behaviors have been characterized as interpersonal communications of pain or suffering. Rate each of the following behaviors as to how regularly this patient exhibits each behavior. Use the following scale to rate each behavior:

1 (Definitely does not exhibit behavior)	2 (Exhibits behavior occasionally)	3 (Exhibits behavior regularly)

1. Facial grimacing. _____
2. Holding or supporting affected body area. _____
3. Question, "Why did this happen to me." _____
4. Shakes affected body area. _____
5. Writhing. _____
6. Distorted gait. _____
7. Frequent shifting of posture or position. _____
8. Indicates unable to engage in certain activities because of pain. _____
9. Requests to be excused from tasks or activities. _____
10. Takes medication (when on a prn schedule). _____
11. Moves extremely slowly. _____
12. Limping. _____
13. Sitting with a rigid posture. _____
14. Movement in a guarded or protective fashion. _____
15. Moaning. _____
16. Uses a heating pad or ice pack. _____
17. Uses a cane, cervical collar, or other prosthetic device. _____
18. Requests help in ambulating. _____
19. Stoops while walking. _____
20. Rubs affected body part. _____
21. Lying down during the day. _____
22. Being irritable. _____
23. Avoidance of physical activity. _____

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Record-Keeping of Pain

Our goal is to more fully understand the nature of your pain. To achieve this I would like you to keep an hourly record of your pain. Attached are ten cards, one for each of ten days. The recrod-keeping is quite easy and straightforward. It consists of rating your pain each hour on a 3 by 5 inch card. On the card you will see, first, the hours of the day are indicated on the top, going from 6 a.m. to 5 a.m. This covers the full 24 hours of one day.

Second, under each hour of the day are numbers from 0 to 5. These refer to the severity of your pain:

5 indicates excruciating, incapacitating, intense pain. The pain is so severe you can do almost nothing else.
4 represents horrible, severe pain attack that makes concentration difficult, but you can do some tasks or something that is undemanding.
3 is distressing, painful but you are able to continue at your job or whatever you were doing.
2 represents discomforting pain, a level that can be ignored at times.
1 represents a low level that enters awareness only at times when attention is devoted to it.
0 represents no pain present.

To summarize:
5 = excruciating, incapacitating, intense, severe
4 = horrible, pain severe, but undemanding tasks possible, concentration difficult
3 = distressing, painful but able to continue job
2 = discomforting, pain can be ignored at times
1 = mild, low level pain only when attended to
0 = none, no pain

I would like you to rate your pain each waking hour of the day. That is, at the end of each hour, think back over the last hour and rate your pain on the 0-to-5 scale. Rate the intensity of the pain you experienced. Was the pain incapacitating? - then circle the 5. Was the pain severe, but you could occupy yourself with undemanding tasks? - then circle the 4. You can likewise use the ratings 0, 1, 2, 3 to describe how the pain affects you.

If you took a pain medication during that hour, in addition to the circle, put an "X" through the number. Thus if your pain was "horrible" and you took a pain medication at 5 PM, you would circle both and put an X through the number 4 under 5 PM.

You should only rate yourself for the waking hours. If you do wake up you can indicate this on the back of the card, where comments are asked for. Under comments, indicate if you got up because of the pain, and the intensity of the pain. In addition, please feel free to make any additional comments about the kinds of pain you are having for example, are they of the dull, aching type, or do they quickly come and then go?

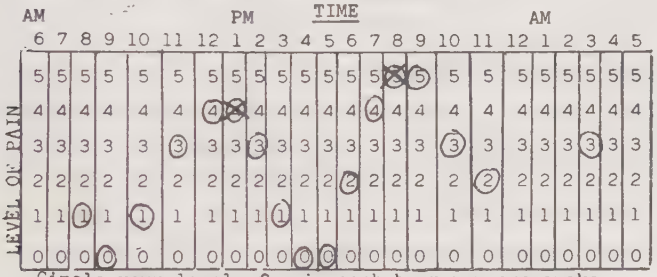
Finally, be sure to put your name on each of the cards, and the dates (Monday, February _____, or whatever day and month it is). You should begin your ratings from 6 AM tomorrow, for the next ten days.

Reproduced below is a sample card, showing how one can rate his pain over a twenty-four hour period. Please note that this is a sample only. Record your pain as you feel it.

There is a key to the severity-of-pain scale on the back of each card. A sample reverse side is also reproduced below, with sample comments on it.

NAME:_____ DATE:_____

Note: KEY and space for comments on back



Circle your level of pain each hour you are awake.
Put an X through the times you take pain medication.

KEY

5 = excruciating, incapacitating, intense, severe
4 = horrible, pain severe, but undemanding tasks possible, concentration difficult
3 = distressing, painful but able to continue job
2 = discomforting, pain can be ignored at times
1 = mild, low level pain only when attended to
0 = none, no pain

COMMENTS:

Awoke briefly at 3:15 a.m. with considerable pain which lasted 15 minutes.

McGill Pain Questionnaire
(Melzack, 1975)

A) Choose <u>one</u> word group that best describes your pain.

_____ Continuous, Steady, Constant

_____ Rhythmic, Periodic, Intermittent

_____ Brief, Momentary, Transient

B) The following words represent pain of increasing intensity

1	2	3	4	5
Mild	Discomforting	Distressing	Horrible	Excruciating

Choose the number of the word that best describes:

_____ Your pain right now

_____ Your pain at its worst

_____ Your pain at its least

_____ The worst toothache you ever had

_____ The worst headache you ever had

_____ The worst stomach-ache you ever had

C) What does your pain feel like?

Some of the following words describe your <u>present</u> pain. Circle the words that best describe it. Leave out any word group that is not suitable. Use only a single word in each appropriate group--the one that applies <u>best</u>.

1	2	3	4
1 Flickering	1 Jumping	1 Pricking	1 Sharp
2 Quivering	2 Flashing	2 Boring	2 Cutting
3 Pulsing	3 Shooting	3 Drilling	3 Lacerating
4 Throbbing		4 Stabbing	
5 Beating		5 Lancinating	
6 Pounding			

5	6	7	8
1 Pinching	1 Tugging	1 Hot	1 Tingling
2 Pressing	2 Pulling	2 Burning	2 Itchy
3 Gnawing	3 Wrenching	3 Scalding	3 Smarting
4 Cramping		4 Searing	4 Stinging
5 Crushing			

9	10	11	12
1 Dull	1 Tender	1 Tiring	1 Sickening
2 Sore	2 Taut	2 Exhausting	2 Suffocating
3 Hurting	3 Rasping		
4 Aching	4 Splitting		
5 Heavy			

13	14	15	16
1 Fearful	1 Punishing	1 Wretched	1 Annoying
2 Frightful	2 Gruelling	2 Blinding	2 Troublesome
3 Terrifying	3 Cruel		3 Miserable
	4 Vicious		4 Intense
	5 Killing		5 Unbearable

17	18	19	20
1 Spreading	1 Tight	1 Cool	1 Nagging
2 Radiating	2 Numb	2 Cold	2 Nauseating
3 Penetrating	3 Drawing	3 Freezing	3 Agonizing
4 Piercing	4 Squeezing		4 Dreadful
	5 Tearing		5 Torturing

RATINGS - LOW BACK PAIN STUDY

Date:_____ Patient:_____

Age: School: History: Prof:

Group: FFD (cm): Surgery: Site:

QUESTIONS:

1. How intense is your pain right now on a scale from 0 = no pain to 10 = horrible, excruciating pain?
2. How tense do you feel right now on a scale from 0 = no tension at all to 10 = extremely tense?
3. How unpleasant was the situation/task you just talked about/completed for you - on a scale from 0 = not at all unpleasant to 10 = very unpleasant?
4. How much effort did it cost you to talk about/perform the situation/task - on a scale from 0 = no effort at all to 10 = very great effort?

		pre	post	comments
Baseline	pain			
	tension			
GSR:	unpleas.			
	effort			
Trial 1	pain			
	tension			
_____	unpleas.			
	effort			
Trial 2	pain			
	tension			
_____	unpleas.			
	effort			
Trial 3	pain			
	tension			
_____	unpleas.			
	effort			
Trial 4	pain			
	tension			
_____	unpleas.			
	effort			

West Haven VAMC

Pain Management Program

Manual for Completion of Assessment Report

This manual is for the completion of the structured Assessment Report for the Pain Management Program. As should be apparent by the comprehensiveness and specificity required by the items, the Assessment Report cannot be written until a complete evaluation has been made.

The manual outline follows that of the checklist on the "Assessment Report". The checklist asks for quantitative evaluation of areas of psychological functioning. The items should be answered via the clinician's best judgement considering the information from both the patient and his or her significant other. It is to be completed with as much care and specificity as possible, following the definitions in this manual.

The area to the right is for the clinical narrative. The location of the items in the narrative should correspond to the checklist information. Paragraphs exceeding the length provided should be completed on a separate sheet. The material that should be included in the clinical narrative is specified in the manual under each topic area, however, in general the narrative should include a fuller discussion of the material in the checklist. Information not specifically requested in the checklist but considered by the clinician to be relevant for a particular patient should also be included in the narrative.

In the checklist the following abbreviations are used: VM = Very Much, Mod. = Moderate, Lit. = Little, NA = Not at All and WNL = Within Normal Limits.

1. History:
   A. Etiology - One of the categories listed below must be chosen. See Pain Syndromes and their Treatments, Turk, 1976 for the location of specific pain syndromes within each grouping.

   1. Musculoskeletal - Rheumatic Disease
   2. Headache
   3. Facial
   4. Neck and Cervicobrachial Neuralgia
   5. Low Back Pain and Sciatica
   6. Visceral Pain of the Chest and Abdomen
   7. Peripheral Vascular Disease
   8. Autonomic Reflex Pain
   9. Nerve Compression Syndrome, Painful Sears, Postherpaetic Neuralgia
   0. Intractable Pain and Cancer

   Only one category may be chosen for the checklist. The primary and current reason for the patient's pain is the one to be rated, (e. g. if the patient has phantom limb pain in an amputated leg that was amputated due to cancer, Autonomic Reflex Pain would be chosen; (See Turk, 1976 88). If a patient presents with more than one type of pain, the clinician must determine the "primary" pain.

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B. Date of Onset - Date of onset should be the date of onset of the primary pain to the nearest month and year.

C. Duration - Duration should be the duration of the primary pain to the nearest month and year.

2. Nature:

Constant - Pain that the patient reports as being always or almost always present (e.g., "It's only absent when I sleep", "It begins every day after breakfast and continues all day") and at a constant level of intensity (a shift of no more than three intensity levels on a 1 to 10 scale).

Fluctuating - Pain that is occasionally present or that changes in intensity level (greater than three intensity levels on a 1 to 10 scale) at various times throughout the day.

Recurrent - Pain that is present not more than once every few days.

The narrative for this section should include the nature of the pain fluctuation, if any (e.g. predictable-unpredictable, day-night differences). Potential discriminative stimuli and/or patient behaviors eliciting pain related to the fluctuations should be noted.

3. Site:

A. Site - Only one of the items listed below may be chosen. Choose the category considered to be the most problematic by the patient.

0 - Head
1 - Upper Back, Shoulders
2 - Lower Back, Legs
3 - Stomach, Pelvic
4 - Arm, Hand
5 - Leg, Feet
6 - Multiple Joint
7 - Chest
8 - Other

The narrative should include all sites considered relevant for clinical intervention. Other properties of the pain, not otherwise requested in this section, should also be described.

B. Spread of site since onset -
Yes - Primary site has shifted in location or spread since onset.
No - Primary site has not changed since onset.

The narrative should address in what manner the site of pain has spread since onset. Whether the patient's report of the nature of the spread is consistent with known neurology (i.e. dermatomes) should also be noted.

4. Variables Affecting Pain:

The clinician should fill in the variable(s) and the extent of their effect. Examples of possible variables are: time of day, physical activity, weather, mood, loud noise, cigarette smoking, coffee drinking, etc.

Very much - The variable and pain alteration correspond almost completely (75% to 100% of the time that the pain occurs).

Moderately - The variable and pain alteration correspond most of the time (50% to 75% of the time that pain occurs).

Little - The variable and pain alteration correspond occasionally (25% to 50% of the time that pain occurs).

Not at All - Variable and pain modification do not correspond at all or do so irregularly or very occasionally (e.g. less than 25% of the time that pain occurs).

The narrative should further discuss in what way these variables affect the patient's pain (e.g., weather - humidity or temperature, and in what direction is the pain affected?).

5. Substance Use:
For each substance in this section note the class of drug (e.g. barbiturate, amphetamine). For alcohol list the type of drink consumed (e.g. beer, hard liquor). If no substance is used in a particular category, write "none". If only one substance in a category is used, write "none" in the "second" category. If more than two substances of any class have been used, code only the two most frequently used substances.

Very much - Daily use or routine use that is at least every other day.

Moderately - Less than routine use every other day or non-routine use but occurring a mean frequency of at least three times per week.

Little - Routine or non-routine use occurring at a mean frequency of more than once, but less than three times per week.

Not at All - Not used at all or non-routine use, occurring a mean frequency of once per week or less (e.g. occasional beer with friends on the weekend).

A. Past - Up to one month ago.

B. Present - Within the past month or sooner if significant change has occurred during this time.

Prescribed - Medication that the patient has a current prescription for, and used for purposes of pain relief.

Over the Counter - Medication bought for purposes of pain reduction that does not require a prescription.

Alcohol - Any beverage containing alcohol.

Illicit - Drugs considered illegal or therapeutic medication requiring a prescription that the patient does not have, or a medication no longer prescribed for the patient.

C. Substance Abuse - Excessive substance usage involving "dependence" or "abuse" as defined by DSM-III. (DSM-III, Substance Use Disorders, pgs. 163-179). Choose the DSM-III diagnosis for all rated areas, or write "none".

The narrative should describe, for each category, frequency and regularity of their use, time of day used, whether the patient perceives its use as problematic and any other information judged to be pertinent by the clinician.

6. Medical Treatments:

A. Surgery - Any surgical procedure requiring local or general anesthetic.

Effectiveness rating - Rate the effectiveness of the most effective surgery considering each of the specified time periods for that surgery only.

Immediate - Up to two months post surgery.

Long Term - More than two months post surgery.

+3 Pain completely eradicated
+2 Substantial pain reduction (i.e. 10% to 50% of previous severity or frequency).
+1 Minor pain reduction (i.e. 50% to 90% of previous severity or frequency).
0 No change in pain (plus or minus 10%)
-1 Minor pain exacerbation (i.e. 10% to 50% increase in severity or frequency).
-2 Substantial pain exacerbation (i.e. 50% to 90% increase in severity or frequency).
-3 Exacerbation in severity of frequency of 100% or more.

B. Physical Therapy - Regular visits to a Physical Therapy Clinic for purposes related to the patient's pain problem.

Effectiveness rating - See definitions for Surgery, 6A.

C. Other Medical Treatments - List other medical or quasi-medical procedures attempted for purposes of pain reduction (e.g. hypnosis, acupuncture, chiropractor). If none, write "none".

Effectiveness rating - See definitions for Surgery, 6A.

The narrative should describe all surgical and medical interventions including types, dates, and outcomes. Potential differing patient coping behaviors, co-varying with differences between immediate and long term effectiveness should also be considered. Be sure to obtain a historical report that is as objective as possible.

7. Vocational:

A. Last grade completed
List grade number through high school (i.e. 1 - 12)
13 - College Freshman
14 - College Sophomore
15 - College Junior
16 - College Senior
17 - Anything Past B.A.

B. Employability - Clinician's judgement in patient's potential for minimally stressful, non-physical, full time employment given his or her current level of functioning (e.g. file clerk, assembly line worker).

C. Employed - Current employment status.

Very much - Full time, (at least 30 hours per week).

Moderate - 15 to 20 hours per week.

Little - 5 to 15 hours per week.

D. Desires employment - Clinician's judgement of patient's motivation for employment (full time or part time) at the present time.

Very much - Patient's self report and history indicate a motivation for employment (e.g. vocational training attempts).

Moderate - Patient reports a strong interest with no recent history to corroborate or patient's behavior indicates some interest in employment.

Little - Patient's reports or behavior indicate a minor interest or interest with ambivalence.

Not at All - No indication of desire for employment.

E. Compensation - Money received by the patient or members of his/her immediate family as a function of the patient not being employed. Specification of percentage pertains to insurance company or VA determined levels.

F. Related to Pain Etiology -

Yes - Compensation is specified in the records to be related to Pain Etiology.

? - Not clearly specified in records or the etiology is unclear.

No - Clearly no relation to pain etiology.

The narrative should include: The nature of previous and/or current employment (i.e. length of employment, type of work) the patient's satisfaction with this; was there a change in his vocational status related to his pain; the patient's desires for the future, how realistic these desires may be and why. The rationale for the determination of "Employability" and "Desires Employment" status should also be indicated. The type of work related activities participated in and the physical activity required of these activities should also be discussed.

8. Work related activities:

A. Activities requiring some vocational skills and/or schedules (e.g. hobbies).

Very much - Patient's current activities encompass a regular schedule and more than 30 hours per week.

Moderate - Patient's current activities encompass 15 to 30 hours per week or a non-regular schedule.

Litte - Patient's current activities encompass 5 to 15 hours per week.

Not at All - Patient's current activities encompass less than 5 hours per week.

9. Domestic

A. Living Situation
1 - With Spouse or "girlfriend/boyfriend"
2 - With Spouse and Children
3 - With Children Alone
4 - With Other Relative or Friend
5 - Alone
6 - Other

B. Length of Marriage - To the nearest 0.5 years. If never married code a zero. If married more than once code the length of the longest marriage.

C. Who will function as the significant other -
1 - Spouse
2 - Child
3 - Patent
4 - Other relative or friend
5 - Other
6 - No significant other

D. Number of previous marriages - Number of previous legal marriages or "living together" situations greater than one year in duration.

E. Non-Commitment to Marriage - Difficulties in marital relationship as inferred from either spouse from all data collected.

Very much - A separation of greater than two weeks duration during the past year due to marital disharmony or clinician judgement that marital disharmony is such that the couple is likely to divorce or separate within the next month or two.

Moderate - A separation period less than two weeks duration during the past year due to marital disharmony or clinician judgement that current marital difficulties may lead to separation in the future if not altered.

Little - No previous separation periods. Some disharmony in marriage but judged by the clinician to not be leading to separation.

Not at All - No marital problems as judged by the clinician.

F. Perceived changes from prior to pain onset -

Very much - Substantial perceived changes on the part of the patient from a history of non-problematic marriage ("not at all") to "very much" as specified above, or perceived changes in the opposite direction.

Moderate - Perceived change from "little" to "very much" or "not at all" to "moderate", as specified above, or perceived changes in the opposite direction.

Little - Perceived change of one level as specified above (e.g. "not at all" to "little").

Not at All - No perceived changes from previous marital situation.

G. Marital Communication Deficit -

Very much - Clinician judged inability of spouses to communicate either content or emotion greater than 50% of the time.

Moderate - Clinician judged inability of spouses to communicate either content or emotion 25% to 50% of the time.

Little - Clinician judged inability of spouses to communicate either content or emotion 10% to 25% of the time.

Not at All - No obvious communication difficulties.

H. Dissatisfaction with marriage - Unhappiness with marital relationship as determined by the self-reports of either spouse.

Very much - Patient or spouse reports extreme unhappiness with marriage. Expresses plans to end marriage or would if not for other circumstance (i.e. children, unacceptable religious practice).

Moderate - Patient or spouse reports some unhappiness with marriage. Dissatisfaction of a severity such that at least one spouse has considered separation or divorce.

Little - Patient or spouse reports little unhappiness with marriage. Neither spouse has considered divorce.

Not at All - Patient and spouse completely satisfied with marriage. A few minor problems may be noted and still be scored in this category.

I. Interference with household chores due to pain -

Very much - The patient completely abstains from all household chores or all except for a very few light duties due to his or her pain (e.g. only puts his/her own clothes away, only cleans his/her own dishes from the table.

Moderate - The patient participates in some household chores but participates in only specific non-physical activities (e.g. only washes dishes, won't make the bed or vacuum the floors).

Little - Abstains from only highly physical activities (i.e. washing windows, fixing the plumbing).

Not at All - Patient does not abstain from participating in any household chores due to his/her pain.

J. Effect on relations with other immediate family members -

Very much - Conflicts due to the patient's pain behavior with at least one immediate family member resulting in non-communication, or frequent (more than three per week) or regularly occurring arguments.

Moderate - Conflicts due to the patient's pain behavior with at least one immediate family member resulting in an average of one to three arguments per week or non-regularly occurring arguments.

Little - Occasional conflicts due to the patient's pain behavior with at least one immediate family member, occurring a mean frequency of less than once per week.

Not at all - No obvious conflicts with immediate family members due to the patient's pain behavior.

K. Effect on relations with extended family members -

Very much - See definition for immediate family members.

Moderate - See definition for immediate family members.

Little - See definition for immediate family members.

Not at All - See definition for immediate family members.

The narrative should describe the nature of any marital disharmony that is noted (e.g. content of arguments, who wins, who loses). If there is a marital communication deficit, it should be more explicitly described (i.e. does one spouse ignore the other, is there an apparent lack of concern). Are the domestic problems related to pain behavior? The judged reality basis of the patient's perceptions of change and any disagreements between family members on any of the above topics should be noted.

Sexual Activity:

A. Frequency per week - Frequency of sexual activity, not necessarily leading to intercourse.

Very much - More than 5 times per week.

Moderate - Three to 5 times per week

Little - One to 3 times per week

Not at all - No reported sexual activity

B. Perceived change in frequency - Differences between the patient's perceived pre and post-pain frequency of sexual activity.

Very much - A reported change of frequency equal to or greater than 5 times per week.

Moderate - A reported change of frequency of 1 to 4 times per week.

Little - A reported change of frequency of 1 to 2 times per week.

Not at All - No reported change in frequency.

C. Perceived change in interest - Difference between the patient's reported pre and post-pain perceptions of his/her libido or reports of current pain status affecting participation in sexual activity resulting in altered interest.

Very much - A reported change in interest from desiring sexual activity 5 or more times per week to 1 or fewer times per week or change in the opposite direction.

Moderate - A reported change of 50% or more in the frequency of interest from desiring sexual activity, up to the requirements meeting "very much".

Little - A reported change of 10% to 50% in the frequency of interest from desiring sexual activity.

Not at All - No reported change in interest or a change in frequency of interest less than 10%.

D. Dissatisfaction with - Patient's report of unhappiness.

Very much - The patient reports extreme unhappiness with sexual activity. Would change other than for pain status.

Moderate - The patient reports moderate amount of unhappiness with sexual activity. Would change other than for pain status but it is not a major source of discontent.

Little - The patient reports little unhappiness when asked. Not a source of discontent.

Not at All - No unhappiness reported. Completely satisfied with sexual activity.

E. Sexual Dysfunction - Patient's report, or information documented in the records of a psychogenic or physiologically based sexual dysfunction.

Very much - The patient is completely unable to participate in sexual activity.

Moderate - The patient's ability to participate in sexual activity is partially affected.

Little - The patient's ability to participate in sexual activity is minimally affected.

Not at All - No sexual dysfunction.

The narrative should address each of the above areas, noting the nature of any reported changes, types of sexual activities that the patient may report an inability to perform and apparent spouse responses. The judged validity of the patient's perception of change should be addressed. The type of sexual dysfunction, the patient's intellectual awareness of, and potential for remediation should also be noted.

11. Social Activities: Activities the patient considers enjoyable to participate in (i.e. recreational, athletic, interpersonal).

A. Perceived change in type since onset - The patient's report of change in type of activities that he/she is participating in from pre to post-pain status.

Very much - The patient reports a major change in the type of social activities (e.g. patient reports he/she previously participated in athletics as a social activity now cannot do this at all).

Moderate - The patient reports a significant change in the type of social activities but continues to participate in less taxing activities or same activities at a decreased frequency.

Little - The patient reports a minor alteration in the type of social activities (e.g. used to play softball, now goes bowling).

Not at All - No reported changes in social activities.

B. Perceived change in frequency since onset - The patient's report of change in frequency of activities he/she is participating in from pre to post-pain status.

Very much - Report of change from daily or regular social participation to less than two times per week.

Moderate - Report of change equivalent to "very much" in a non-primary social activities or smaller but significant change in primary social activities.

Little - Report of small but significant change in non-primary social activities or minor change in primary activities.

Not at All - No change in primary social activites or small change in non-primary social activities.

C. Perceived change in interest since onset - Reported change in patient's preferred primary social activities (not necessarily what he/she is doing but what he/she would like to do).

Very much - Shift from one form of activity to another (e.g. from athletics to socializing at a bar).

Moderate - Non-complete shift of form of activities (i.e. change in what patient would like to spend most of his/her time doing but continues to desire participation in previous preferred activity at a decreased frequency or lesser capacity).

Little - Shift of preference within one class of activity, (e.g. softball to bowling) or modification of non-primary interests.

Not at All - No change in interest or small shift within one class of non-primary activity.

D. Dissatisfaction with -

Very much - Patient reports extreme unhappiness with current social activities. Would change but cannot due to pain status.

Moderate - Patient reports significant unhappiness with social activities. Patient probably would change or it is not clear if patient would change due to pain status.

Little - Patient reports small amount of unhappiness. Change in pain status would not have a significant effect.

Not at All - No unhappiness reported. Completely satisfied with social activities.

The narrative should address each of the above areas, noting the nature of any reported changes, types of social activities (pre and post), frequency, possible reasons for any changes noted and spouse responses. The judged validity of the patient's perception of change should be addressed.

12. Neurovegetative Signs:

A. Sleep problems - Difficulty falling asleep, sleep continuity disturbance, early morning awakening or excessive sleeping.

Very much - At least one of sleep problems 5 or more times per week.

Moderate - At least one of sleep problems 3 to 5 times per week.

Little - At least one of sleep problems 2 to 3 times per week.

Not at All - No sleep problems or a frequency of less than 2 times per week.

B. Appetite Problems - Increase or decrease in appetite or weight.

Very much - Significant appetite problem (e.g. one meal per day or less) or continuous or frequent binge eating) or weight loss or increase greater than 20% in past three months that is not due to dieting.

Moderate - Appetite reduction (or increase) of 10% or greater in past 3 months not due to a diet, or report of significantly affected diet, but less than meeting the criteria for "very much".

Little - Report of appetite reduction (or increase) of 5% to 10% in past three months not due to a diet or report of occasionally affected diet (less than daily).

Not at all - No appetite or weight changes in past three months not related to diet.

C. Urination Problems -

Very much - Extreme pain or discomfort urinating due to the patient's pain problem. Pain of a severity that the patient avoids or delays urinating as much as possible.

Moderate - Moderate pain or discomfort in urinating due to the patient's pain problem. The patient does not usually avoid or delay urinating.

Little - Mild discomfort when urinating due to the patient's pain problem. Never avoids or delays urinating due to this.

Not at All - No problem noted.

D. Bowel Movement Problems -

Very much - See description for urination.

Moderate - See description for urination.

Little - See description for urination.

Not at All - See description for urination.

The narrative should describe the nature of each problem noted, how the patient has attempted to deal with this in the past (i.e. behaviorally, medically) and the outcomes. Whether the above noted neurovegetative problems are directly related to pain or to some other variable (e.g. medication side effect) should also be noted.

Mental Status:

Only "mood" should be coded, however, a short mental status evaluation should be completed and placed in the narrative including: general appearance and behavior; speech rate and pattern, presence of signs of thought disorder, general affect, obvious presence of concentration or memory difficulties.

Mood - The clinician's judgement of the patient's mood via the patient's self-report, recent history and behavioral observation.

Very affected - Extremely despondent or anxious. Patient may report or clinician may observe frequent tearfulness, pacing (but not due to a physical pain behavior), no sense of humor, anger. The patient reports seriously considered suicidal ideation in past few months.

Moderately affected - The patient reports significant depression, anxiety or anger. Occasional tearfulness reported (approximately once per week). No serious suicidal ideation reported.

Slightly affected - The patient reports infrequent sadness or crying (crying episodes not regular, or less than once per week). Mild agitation or anger noted.

Within normal limits - Mood is not noted to be depressed or anxious.

Coping Skills:

Any behavior the patient reports performing or is observed to do that is judged by the clinician to be for the purposes of pain reduction or management, (i.e. rubbing, shifting on seat, distracting himself or herself).

A. Physical - Any coping behaviors consisting primarily of physical movements.

Very much - Constant or almost constant movement or display (more than 76% of the time pain is reported) of a physical coping skill.

Moderate - Fifty to 75% of a physical coping skill when pain is reported.

Little - Ten to 50% display of a physical coping skill when pain is reported.

Not at All - Less than 10% display of a physical coping skill when pain is reported.

B. Cognitive - Any coping behavior the patient is observed to emit or reports that is primarily cognitive.

Very much - See description for physical coping skills.

Moderate - See description for physical coping skills.

Little - See description for physical coping skills.

Not at All - See description for physical coping skills.

The narrative should address the type of coping skills that are utilized, under what circumstances and their outcomes. How these particular coping skills were arrived at and a history of previous coping skills if available should also be discussed.

15. Current Psychological Treatment:

Yes - Currently seeing a mental health worker or at least on a semi-regular basis. May be for pain or non-pain related issues.

No - Not currently seeing a mental health worker.

A. Problem - Describe as specifically as possible. Use of DSM-III terminology is recommended if this will specify the problem (e.g. Major Depressive Episode, Social Phobia).

B. Treatment - Description of treatment should be as specific as possible via clinician's judgement of patient's description (e.g. systematic desensitization, family therapy, insight oriented therapy).

The narrative should describe the nature of the problem the person is currently in treatment for, the type of treatment being received and other relevant information.

16. Previous Psychological Treatment:

Very much - A history of regular visits to a mental health professional of more than 6 months duration.

Moderate - A history of regular visits to a mental health professional of more than 2 months, but less than 6 months duration.

Little - From 3 visits to 2 months of regular visits to a mental health worker.

Not at All - A history of not more than 2 visits to a mental health worker.

A. Problem - See current Psychological Treatment, Problem, 11A

B. Treatment - See current Psychological Treatment, Treatment, 11B

C. Effectiveness Rating

+3 - Problem completely eradicated

+2 - Substantial symptom reduction (i.e. 10% to 50% of previous severity or frequency).

+1 - Minor symptom reduction (i.e. 50% to 90% of previous severity or frequency).

0 - No change in symptoms (plus or minus 10%)

-1 - Minor symptom exacerbation (i.e. 10% to 50% increase in severity or frequency).

-2 - Substantial symptom exacerbation (i.e. 50% to 90% increase in severity or frequency).

-3 - Exacerbation in severity or frequency of 100% or more.

The narrative should describe in detail the patient's history of psychological treatments. Specific information should include: the nature of the problems the person sought help for; their possible relationship to his/her pain or pain etiology; the number of practitioners seen and more extensive information about the type of treatment(s) given.

Recommendations:

The clinician should code his recommendation. If the patient is rejected, the narrative should state the rationale for this decision as well as any other pertinent information.

WEST HAVEN VAMC
PAIN MANAGEMENT PROGRAM
ASSESSMENT REPORT

Clinician_____

Name & PMP _____(1-3) Date_____

Referred by_____ Age_____(4-5) Sex_____(6)

Narrative

1. History:
   A. Etiology (see manual)_____ (7)
   B. Date of Onset _____
   C. Duration (years)_____ (8-9)

(if you need more room attach another sheet)

2. Nature: (check one)
   Constant (0) _____
   Fluctuating (1) _____
   Recurrent (2) _____ (1o)

(same as above)

3. Site:
   A. Site _____ (11)
   B. Spread of site since onset
      Yes ____ No_____ (12)

(same as above)

4. Variables Affecting Pain: (specify)

   _____
   VM__Mod.__Lit.__NA__ (13)

   _____
   VM__Mod.__Lit.__NA__ (14)

   _____
   VM__Mod.__Lit.__NA__ (15)

   _____
   VM__Mod.__Lit.__NA__ (16)

(same as above)

Substance Use:

A. Substance Use: Past

Prescribed_____

VM___Mod.__Lit.__NA__ (17)

Prescribed_____

VM___Mod.__Lit.__NA__ (18)

Over the Counter_____

VM___Mod.__Lit.__NA__ (19)

Over the Counter_____

VM___Mod.__Lit.__NA__ (2o)

Alcohol_____

VM___Mod.__Lit.__NA__ (21)

Alcohol_____

VM___Mod.__Lit.__NA__ (22)

Illicit_____

VM___Mod.__Lit.__NA__ (23)

Illicit_____

VM___Mod.__Lit.__NA__ (24)

B. Substance Use: Present

Prescribed_____

VM___Mod.__Lit.__NA__ (25)

Prescribed_____

VM___Mod.__Lit.__NA__ (26)

Over the Counter_____

VM___Mod.__Lit.__NA__ (27)

Over the Counter_____

VM___Mod.__Lit.__NA__ (28)

Alcohol _____

VM___Mod.__Lit.__NA__ (29)

VM___Mod.__Lit.__NA__ (3o)

Illicit_____

VM___Mod.__Lit.__NA__ (31)

Illicit_____

VM___Mod.__Lit.__NA__ (32)

(if you need more room attach another sheet)

C. Substance Abuse: Past

Alcohol_____

Dependence_____Abuse_____ (33)

Illicit_____

Dependence_____Abuse_____ (34)

D. Substance Abuse: Present

Alcohol_____

Dependence_____Abuse_____ (35)

Illicit_____

Dependence_____Abuse_____ (36)

(if you need more room attach another sheet)

6. Medical Treatments

A. Surgery

4 or more___1-3___0____ (37)

Effectiveness Rating (-3 to 3)

Immediate_____ (38-39)

Long Term_____ (4o-41)

B. Physical Therapy

Yes_____ No_____(42)

Effectiveness Rating (-3 to 3)

Immediate_____(43-44)

Long Term_____(45-46)

C. Other Medical Treatments

Effectiveness Rating (-e to 3)

Immediate_____(47-48)

Long Term_____(49-5o)

_____

Effectiveness Rating (-3 to 3)

Immediate _____(51-52)

Long Term_____(53-54)

7. Vocational

A. Last Grade Completed___(55-56)

same as above)

Narrative

B. Employability
VM___Mod.___Lit.___NA___ (57)

C. Employed
VM___ Mod.___Lit.___NA___ (58)

D. Desires Employment
VM___Mod.___Lit.___NA___ (59)

E. Compensation_____% (6o)

F. Related to Pain Etiology
Yes_____?_____No_____ (61)

(if you need more room attach another shee

Work Related Activities:

_____
VM___Mod.___Lit.___NA___(62)

(same as above)

Domestic

A. Living Situation (see manual)_____(63)

B. Length of Marriage (years)_____(64-65)

C. Who will function as significant other?
_____(66)

D. Number of previous marriages_____(67)

E. Non-Commitment to Marriage
VM___Mod.___Lit.___NA___(68)

F. Perceived change from prior to pain onset
VM___Mod.___Lit.___NA___(69)

G. Marital Communication Deficit
VM___Mod.___Lit.___NA___(7o)

H. Disssatisfaction with marriage
VM___Mod.___Lit.___NA___(71)

I. Interference with household chores
VM___Mod.___Lit.___NA___(72)

J. Effect on relations with other immediate family members
VM___Mod.___Lit.___NA___(73)

K. Effect on relations with extended family members
VM___Mod.___LIt.___NA___(74)

(same as above)

0. Sexual Activity

A. Frequency per week_____ (75)

B. Perceived change in frequency since onset

VM___Mod.__Lit.___NA___ (76)

C. Perceived change in interest since onset

VM___Mod.__LIt.___NA___ (77)

D. Dissatisfaction with

VM___Mod.__Lit.___NA___ (78)

E. Sexual Dysfunction

VM___Mod.__Lit.___NA___ (79)

(if you need more room attach another sheet)

1. Social Activities

A. Perceived change in type since onset

VM___Mod.__Lit.___NA___ (8o)

B. Perceived change in frequency since onset

VM___Mod.___Lit.___NA___ (81)

C. Perceived change in interest since onset

VM___Mod.___Lit.___NA___ (82)

D. Dissatisfaction with

VM___Mod.___LIt.___NA___ (83)

(same as above)

2. Neurovegetative Signs

A. Sleep Problem

VM___Mod.___Lit.___NA___ (84)

B. Appetite Problems

VM___Mod.___Lit.___NA___ (85)

C. Urination Problems

VM___Mod.___Lit.___NA___ (86)

D. Bowel Movement Problems

VM___Mod.___Lit.___NA___ (87)

(same as above)

3. Mental Status: (see manual)

Mood -

Very affected___Mod.affected_____

Sligthly affected_____WNL_____ (88)

(if you need more room attach another sh

4. Coping Skills

A. Physical

VM___Mod.__Lit.__NA___ (89)

B. Cognitive

VM___Mod.__Lit.__NA___ (9o)

(same as above)

15. Current Psychological Treatment

Yes_____No____ (91)

A. Problem

B. Treatment

(same as above)

16. Previous Psychological Treatment

VM___Mod.___Lit.__NA____ (92)

A. Problem!_____

B. Treatment_____

C. Effectiveness Rating (-3 to 3)____(93-94)

(same as above)

17. Recommendations

Accept_____Reject____ (95)

(same as above)

SUMMARY:

Name: _____ Date: _____

Self-Analysis Questionnaire

Directions: A number of statements which people have used to describe themselves are given below. Read each statement and then circle the appropriate number to the right of the statement to indicate how you feel right now, that is, at this moment.

There are no right or wrong answers. Do not spend too much time on any one statement but give the answer which seems to describe your present feelings best.

	Not at All	Somewhat	Moderately So	Very Much So
1. I feel calm.....	1	2	3	4
2. I feel secure.....	1	2	3	4
3. I am tense.....	1	2	3	4
4. I feel regretful.....	1	2	3	4
5. I feel at ease.....	1	2	3	4
6. I feel upset.....	1	2	3	4
7. I am presently worrying over possible misfortunes.....	1	2	3	4
8. I feel rested.....	1	2	3	4
9. I feel anxious.....	1	2	3	4
10. I feel comfortable.....	1	2	3	4
11. I feel self-confident.....	1	2	3	4

	Not at All	Somewhat	Moderately So	Very Much So
12. I feel nervous.....	1	2	3	4
13. I am jittery.....	1	2	3	4
14. I feel "high strung".....	1	2	3	4
15. I am relaxed.....	1	2	3	4
16. I feel content.....	1	2	3	4
17. I am worried.....	1	2	3	4
18. I feel over-excited and "rattled".....	1	2	3	4
19. I feel joyful.....	1	2	3	4
20. I feel pleasant.....	1	2	3	4

ADJECTIVE CHECKIST

Name_____Age_____Sex_____

Date_____Highest grade completed in school_____

DIRECTIONS: Below you will find words which describe different kinds of moods and feelings. Check the words which describe How You Feel Now -- Today. Some of the words may sound alike, but we want you to check all the words that describe your feelings. Work rapidly and check all of the words which describe how you feel today.

1. _____ Sorrowful
2. _____ Lively
3. _____ Uneasy
4. _____ Tormented
5. _____ Low-spirited
6. _____ Clean
7. _____ Discouraged
8. _____ Suffering
9. _____ Broken-hearted
10. _____ Easy-going
11. _____ Downhearted
12. _____ Washed Out
13. _____ Playful
14. _____ Joyless
15. _____ Despairing
16. _____ Gay
17. _____ Friendly
18. _____ Successful
19. _____ Rejected
20. _____ Crestfallen
21. _____ Jolly
22. _____ Deserted
23. _____ Grieved
24. _____ Low
25. _____ Steady
26. _____ Wretched
27. _____ Terrible
28. _____ Inspired
29. _____ Woeful
30. _____ Unworthy
31. _____ Joyous
32. _____ Destroyed
33. _____ Somber
34. _____ Unconcerned

NAME _____ B - 8 DATE _____

BECK INVENTORY

1. 0 I do not feel sad.
   1 I feel sad.
   2 I am sad all the time and I can't snap out of it.
   3 I am so sad or unhappy that I can't stand it.

2. 0 I am not particularly discouraged about the future.
   1 I feel discouraged about the future.
   2 I feel I have nothing to look forward to.
   3 I feel that the future is hopeless and that things cannot improve.

3. 0 I do not feel like a failure.
   1 I feel I have failed more than the average person.
   2 As I look back on my life, all I can see is a lot of failures.
   3 I feel I am a complete failure as a person.

4. 0 I get as much satisfaction out of things as I used to.
   1 I don't enjoy things the way I used to.
   2 I don't get real satisfaction out of anything anymore.
   3 I am dissatisfied or bored with everything.

5. 0 I don't feel particularly guilty.
   1 I feel guilty a good part of the time.
   2 I feel quite quilty most of the time.
   3 I feel guilty all of the time.

6. 0 I don't feel I am being punished.
   1 I feel I may be punished.
   2 I expect to be punished.
   3 I feel I am being punished.

7. 0 I don't feel disappointed in myself.
   1 I am disappointed in myself.
   2 I am disgusted with myself.
   3 I hate myself.

8. 0 I don't feel I am any worse than anybody else.
   1 I am critical of myself for my weaknessess or mistakes.
   2 I blame myself all the time for my faults.
   3 I blame myself for everything bad that happens.

9. 0 I don't have any thoughts of killing myself.
   1 I have thoughts of killing myself, but I would not carry them out.
   2 I would like to kill myself.
   3 I would kill myself if I had the chance.

10. 0 I don't cry any more than usual.
    1 I cry more now than I used to.
    2 I cry all the time now.
    3 I used to be able to cry, but now I can't cry even though I want to

11. 0 I am no more irritated now than I ever am.
    1 I get annoyed or irritated more easily than I used to.
    2 I feel irritated all the time now.
    3 I don't get irritated at all by the things that used to irritate me

12. 0 I have not lost interest in other people.
1 I am less interested in other people than I used to be.
2 I have lost most of my interest in other people.
3 I have lost all of my interest in other people.

13. 0 I make decisions about as well as I ever could.
1 I put off making decisions more than I used to.
2 I have greater difficulty in making decisions than before.
3 I can't make decisions at all anymore.

14. 0 I don't feel I look any worse than I used to.
1 I am worried that I am looking old or unattractive.
2 I feel that there are permanent changes in my appearance that make me look unattractive.
3 I believe that I look ugly.

15. 0 I can work about as well as before.
1 It takes an extra effort to get started at doing something.
2 I have to push myself very hard to do anything.
3 I can't do any work at all.

16. 0 I can sleep as well as usual.
1 I don't sleep as well as I used to.
2 I wake up 1-2 hours earlier than usual and find it hard to get back to sleep.
3 I wake up several hours earlier than I used to and cannot get back to sleep.

17. 0 I don't get more tired than usual.
1 I get tired more easily than I used to.
2 I get tired from doing almost anything.
3 I am too tired to do anything.

18. 0 My appetite is no worse than usual.
1 My appetite is not as good as it used to be.
2 My appetite is much worse now.
3 I have no appetite at all anymore.

19. 0 I haven't lost much weight, if any, lately.
1 I have lost more than 5 pounds.
2 I have lost more than 10 pounds.
3 I have lost more than 15 pounds.
I am purposely trying to lose weight by eating less. Yes____ No____

20. 0 I am no more worried about my health than usual.
1 I am worried about physical problems such as aches and pains; or upset stomach; or constipation.
2 I am very worried about physical problems and it's hard to think of much else.
3 I am so worried about my physical problems, that I cannot think about anything else.

21. 0 I have not noticed any recent change in my interest in sex.
1 I am less interested in sex than I used to be.
2 I am much less interested in sex now.
3 I have lost interest in sex completely.

Name:_____ Date:_____

Pain Experience Questionnaire

Many people report having the following kinds of thoughts and feelings when their pain is severe. We would like to know which of these thoughts and feelings you experience when your pain is severe and to what extent you experience them. Please indicate the extent to which each of the following statements is representative of your thoughts and feelings by circling the appropriate number, ranging from "0" for "not at all" to "6" for "very much".

	Not at All						Very Much
1. I tell myself to stay in control...	0	1	2	3	4	5	6
2. I feel sorry for myself.....	0	1	2	3	4	5	6
3. I worry about by family.....	0	1	2	3	4	5	6
4. I feel helpless.....	0	1	2	3	4	5	6
5. I feel afraid that my pain will get worse.....	0	1	2	3	4	5	6
6. I am depressed because of my pain..	0	1	2	3	4	5	6
7. I feel overwhelmed.....	0	1	2	3	4	5	6
8. I feel I don't want to upset anyone by telling them how bad the pain is.....	0	1	2	3	4	5	6
9. I wonder what it would be like to never have pain.....	0	1	2	3	4	5	6
10. I think, "Things could be worse"...	0	1	2	3	4	5	6
11. I worry about future plans.....	0	1	2	3	4	5	6
12. I think, "I hate to take medicine but it looks like I will have to this time".....	0	1	2	3	4	5	6
13. I feel dependent.....	0	1	2	3	4	5	6
14. I feel anxious.....	0	1	2	3	4	5	6
15. I think this is a challenge.....	0	1	2	3	4	5	6
16. I think, "Why me? Why do I have this pain?"	0	1	2	3	4	5	6
17. I think, "all I can do is wait it out".....	0	1	2	3	4	5	6
18. I wonder how long this will last...	0	1	2	3	4	5	6

	Not at All						Very Much
19. I wonder if they will ever find a cure for my pain.....	0	1	2	3	4	5	6
20. I feel disappointed with myself for giving in to the pain.....	0	1	2	3	4	5	6
21. I think, "This pain is driving me crazy".....	0	1	2	3	4	5	6
22. I think about my pain getting worse..	0	1	2	3	4	5	6
23. I feel irritable	0	1	2	3	4	5	6
24. I feel panic stricken.....	0	1	2	3	4	5	6
25. I think people should be more considerate.....	0	1	2	3	4	5	6
26. I am determined not to take any pain killers.....	0	1	2	3	4	5	6
27. I think of nothing other than my pain.....	0	1	2	3	4	5	6
28. I feel impatient with everyone.....	0	1	2	3	4	5	6
29. I think to myself, "I can take it"..	0	1	2	3	4	5	6
30. I feel that I am being punished.....	0	1	2	3	4	5	6
31. I think, "When I feel like this, I need comfort from my family and friends".....	0	1	2	3	4	5	6
32. I feel angry.....	0	1	2	3	4	5	6
33. I think, "It is so hard to do anything when I have pain".....	0	1	2	3	4	5	6
34. I think everyone should be quiet and leave me alone.....	0	1	2	3	4	5	6
35. I feel frustrated.....	0	1	2	3	4	5	6
36. I think of others who are worse off than me.....	0	1	2	3	4	5	6
37. I feel everyone is getting on my nerves.....	0	1	2	3	4	5	6
38. I wonder why my pain is worse now....	0	1	2	3	4	5	6
39. I feel guilty about having pain.....	0	1	2	3	4	5	6
40. I think about whether life is worth living.....	0	1	2	3	4	5	6

MHLC HEALTH QUESTIONNAIRE

Form A

Please answer the following questions by circling the number of the response that most clearly reflects how you feel.

1 = Strongly disagree
2 = Moderately disagree
3 = Slightly disagree
4 = Slightly agree
5 = Moderately agree
6 = Strongly agree

1. If I get sick, it is my own behavior which determines how soon I get well again..... 1 2 3 4 5 6

2. No matter what I do, if I am going to get sick, I will get sick..... 1 2 3 4 5 6

3. Having regular contact with my physician is the best way for me to avoid illness..... 1 2 3 4 5 6

4. Most things that affect my health happen to me by accident. 1 2 3 4 5 6

5. Whenever I don't feel well, I should consult a medically trained professional..... 1 2 3 4 5 6

6. I am in control of my health..... 1 2 3 4 5 6

7. My family has a lot to do with my becoming sick or staying healthy..... 1 2 3 4 5 6

8. When I get sick I am to blame..... 1 2 3 4 5 6

9. Luck plays a big part in determining how soon I will recover from an illness..... 1 2 3 4 5 6

10. Health professionals control my health..... 1 2 3 4 5 6

11. My good health is largely a matter of good fortune..... 1 2 3 4 5 6

12. The main thing which affects my health is what I myself do. 1 2 3 4 5 6

13. If I take care of myself, I can avoid illness..... 1 2 3 4 5 6

14. When I recover from an illness, it's usually because other people (for example, doctors, nurses, family, friends) have been taking good care of me..... 1 2 3 4 5 6

15. No matter what I do, I'm likely to get sick..... 1 2 3 4 5 6

16. If it's meant to be, I will stay healthy..... 1 2 3 4 5 6

17. If I take the right actions, I can stay healthy..... 1 2 3 4 5 6

18. Regarding my health, I can only do what my doctor tells me to do..... 1 2 3 4 5 6

Marital Adjustment Scale

Name _____ Spouse _____

Please complete the following:

1. Circle the dot on the scale line below which best describes the degreee of happiness, everything considered, of your present marriage. The middle point, happy, represents the degreee of happiness which most people get from marriage, and the scale gradually ranges from one side of those few who are very unhappy in marriage, and on the other, to those few who experience extreme joy or felicity in marriage.

0 2 7 15 20 25 35

. . . . . .

Very Unhappy — Happy — Perfectly Happy

State the approximate extent of agreement or disagreement between you and your mate on the following items. Please check each row.

	Always Agree	Almost Always Agree	Occasionally Disagree	Frequently Disagree	Almost Always Disagree	Always Disagree
2. Handling family finances						
3. Matters of Recreation						
4. Demonstration of Affection						
5. Friends						
6. Sex Relations						
7. Conventionality (Right, good, or proper conduct)						
8. Philosophy of life						
9. Ways of Dealing with In-Laws						

For the following questions , check one alternative.

10. When disagreements arise, they usually result in:
husband giving in ____wife giving in _____
agreement by mutual give and take ____

11. Do you and your mate engage in outside interests together?
all of them ____ some of them____ very few of them _____
none of them ____

12. In leisure time, do you generally prefer:
to be "on the go"_____ to stay at home _____
Does your mate generally prefer:
to be "on the go"_____ to stay at home _____

13. Do you ever wish you had not married?
frequently ____ occasionally _____ rarely ____ never _____

14. If you had to live your life over, do you think you would:
marry the same person _____ marry a different person _____
not marry at all _____

15. Do you confide in your mate?
almost never _____ rarely _____ in most things _____
in everything _____

Information about :

Psychophysiological Assessment of Chronic Pain

You are invited to participate in a study examining the relationship of stress, muscle tension, and chronic pain. It has recently been found that much of the chronic pain patients experience comes from chronically elevated muscle tension in addition to other problems. This study attempts to examine this relationship in greater detail.

The procedure to be used will involve the measurement of your muscle tension, heart-rate and perspiration while you engage in a period of rest (10 minutes), 2 common and simple mental tasks, and talk about 2 recent experiences. Each of these tasks will have the same structure: for 1 minute you will be asked to rest, for 1 minute you will perform the task, and for 5 minutes after each task you will rest again. To measure your body signals during the rest and task periods , sensors will be attached to your back, forehead, site of pain, and your fingers (after the skin has been cleansed with alcohol), while you are sitting in a comfortable chair. The measurement of these signals is non-painful and harmless, and there will be no electricity passed to your body. The method is similar to taking an electrocardiogram which you might have previously experienced. The sensors may cause slight irritation of your skin, which usually quickly disappears after their removal. Taking off the sensors may be a little bit painful if you have hair at the site of the attachment - it is like taking off a band-aid.

The entire procedure will last about 90 minutes. Before and after the tasks you will be asked to estimate your level of tension, pain, the unpleasantness of the situation, and the effort it took to complete the task. At the beginning and the end of the assessment we will ask you to fill out a brief questionnaire about your present mood. If you are a pain patient , but not participant of the Pain Management Program, you will be briefly interviewed about the history of your pain problem, and you will be asked to fill out 3 questionnaires about your pain and your health at the end of the session.

If you are a patient Of the Pain Management Program we will ask you to return for a second assessment at the end the end of your treatment. This is, however, completely voluntary and not part of the treatment.

There are no known risks involved in this measurement . Sitting still for more than one hour and participation in the slightly stressful tasks may, however, become uncomfortable for some pain patients, and may aggravate their pain. If you think that you do not want to take this risk, feel free to decline participation in this assessment. You may also terminate your participation any time during the study.

This assessment is not a treatment for your pain problem, nor are there any direct benefits involved if you participate in the study. The data gained are, however, part of the comprehensive evaluation in the Pain Management Program and may be useful for planning the most effective treatment possible. This study will help us gain knowledge about chronic pain patients in general, and to develop more effective assessment and treatment strategies for the future. We will be happy to inform you in greater

Continuation of the Information Sheet

detail about the purpose of this study at the end of your assessment.

If you have any questions at any time concerning this procedure, we will be happy to answer them. You are free to decline entrance into or withdraw your participation in this study at any time. Such refusal or withdrawal will not affect your care or treatment in any way.

If you should be physically injured as a result of your participation in the study, you would receive medical care and treatment for such injuries. If you are a veteran, you might be entitled to compensation under 38 U.S.C. 310 or 351. Whether or not you are a veteran you might also be entitled to recover under the Federal Tort Claims Act.

If you should suffer any physical injury as a result of your participation in this study, please contact Medical Administration Service where the matter will be reviewed with you and you will be given further information as to how to proceed.

_____ _____

Date Subject

_____

Witness: Name and Address

_____ _____

Signature of Witness Investigator (Signature)

INSTRUCTIONS FOR THE BASELINE

Please sit back in your chair with your feet positoned on the floor, your arms on the armrest, and your head at the headrest, and make yourself comfortable. All I want you to do for the next 10 minutes is to sit still and rest. It is important that you try not to move, sneeze or cough, since all movements will disturb the recordings. You should, however, not get upset if you can absolutely not avoid a movement. These disturbances of the signal can be eliminated later on, if necessary. Please keep your eyes open and try to move them as little as possible, since gross eyemovements will also influence the recordings.
Just make yourself comfortable and rest for 10 minutes as soon as I tell you the beginning of the recording period. At the end of the 10 minutes a beeper will signal the end of the recordings and I will also tell you that the 10 minutes are over. Then you'll be free to move and talk again. Do you still have any questions? ...Okay, we'll start <u>now</u>...

INSTRUCTIONS FOR THE EXPERIMENTAL CONDITIONS

1. BEFORE THE PRETRIAL BASELINE

All I want you to do now is to sit back in your chair just as you sat before and rest again for one minute just as you did before. Please keep your eyes open and try to move as little as possible. After one minute the beeper will sound and you will then receive instructions for the task that will follow. You will be asked to perform this task for one minute and then you will be asked to rest again for 5 minutes. Then a new sequence of one minute rest - one minute task performance and 5 minutes rest will follow. Now let's go through this sequence again...(repeat instruction with patient)...Okay, so all you have to do now is to sit still with your eyes open and rest for one minute until you hear the beeper.

2. BEFORE THE EXPERIMENTAL CONDITIONS

Now, we'll begin with the first task:

(1) ALPHABET

All you need to do is to recite the alphabet for one minute. You can take all the time you want to use. The sole purpose of the task is to see what happens when you perform simple talking. Should you be through with the alphabet before the minute is over, just repeat the alphabet until you hear the sound of the beeper. Do you think that you can do the task? (If the patient says 'no': Well, then how about counting from 1 to 3, and repeating it over until you hear the beeper? All I want is to hear you talk for a minute...).

proceed to 3. Continuation of the task instructions

(2) PAIN

Okay, what I'd like you to do now is to go back in your mind to the last severe pain episode you have had, maybe today, yesterday, or some time this week. It should have a rating of at least 5 on a 10-point scale ranging from no pain to horrible, excruciating pain. If you cannot find a recent pain experience, you may go further back. ...Did you find something? Good. ...What I would

like you to do once I'll give the signal is to talk about this pain experience for one minute. The way I would like you to do it is this: Just really get back into the experience. Describe where you were when the pain started, what you were doing, who else was present, re-experience the pain - what did it feel like, what were you feeling, thinking, doing - just really re-experience the pain as vividly as you can...

proceed to 3. Continuation of the task instructions

(3) STRESS

What I'd like you to do now is to go back in your mind to the last week. Try to remember an event that was upsetting for you, something that made you sad, anxious, angry, something that worried you. Just any unpleasant situation that would get a rating of at least 5 on a scale from 0 = not at all stressfull to 10 = extremely stressfull... Did you find something? You may go further back in time if you can't find anything that upset you very much last week... Good. What I'd like you to do once I'll give the signal is to talk about this upsetting event for one minute. The way I'd like you to do that is this: Just really try to get back into the experience. Describe where you were, when you became so upset, if anyone else was involved in it, what you and the other persons did, what exactly made you so upset, what you thought and felt in the situation, how you reacted to it - just re-experience the event as vividly as possible...

proceed to 3. Continuation of the task instructions

(4) MENTAL ARITHMETIC

What I'd like you to do next is to start at the number 758 and count backwards by steps of 7 as fast as you can. You should do this for one minute until the beeper signals the end of the task. Do you think you can do this? (If the person signals that he or she is not sure if he or she can do the task , the instruction continues)... Well, this task is not supposed to be easy. Do you think that you can at least try it or are you sure that you cannot do it at all? ...Let me give you an example: If you'd start at 300, you'd go: 293, 286, 279, and so forth. Do you think you could do the same starting at 758? ...(If the subject still

signals no, the instruction continues)...How about going backwards in steps of 3...(If the subject still signals inability) ...How about counting backwards from 100 in steps of 3...(If still unable)...How about counting backwards in steps of 1?

3. CONTINUATION OF THE TASK INSTRUCTIONS

...Now, it is important that you don't begin with the task before I tell you to start and it is also very important that you try not to move while you are talking. People tend to move their head, hands, legs and so forth when talking. But please try to avoid it anyway. After you have talked for one minute a beeper will sound again signaling you to stop whatever you are doing. Please stop the task right on the sound of the beeper. Do not try to go on. It is very important that you stop whatever you are doing right on the signal because a clock starts counting when the beeper sounds. If you continue with your task beyond the signal, the values will be wrong. So please stop talking on the signal but do not yet get up. I'd like you to remain seated in your chair and rest again for 5 more minutes. This will help us to determine how long it takes for your measures to recover from talking. Therefore you should just sit and rest during these 5 minutes without moving or talking. The beeper will signal you the end of this period and then you'll be able to move or talk again.

So the sequence is (repeat with patient): When I give the signal you do your task for one minute - when you hear the beeper you stop right there - then you'll rest for 5 minutes until you hear the beeper again. Then the task will be over and we can start a new sequence. Do you have any questions? ...Good, then start now...

Means and Standard Deviations of the Physiological Variables in the ALPHABET Condition

	Chronic Back Pain		General Pain		Healthy Controls	
	M	(SD)	M	(SD)	M	(SD)
Left Erector Spinae EMG (in Microvolts):						
- Pretrial Baseline	2.744	(4.886)	1.178	(.914)	1.210	(.666)
- Trial	3.088	(4.032)	1.594	(1.294)	1.410	(.586)
- Posttrial Baseline	2.819	(4.628)	1.298	(1.249)	1.263	(.801)
Delay in Return to Baseline (in seconds):	20.588	(44.825)	18.353	(52.599)	27.059	(71.478)
Right Erector Spinae EMG (in Microvolts):						
- Pretrial Baseline	1.082	(1.456)	1.856	(1.165)	1.390	(.659)
- Trial	2.216	(1.329)	2.115	(1.334)	1.600	(.564)
- Posttrial Baseline	1.760	(.995)	1.832	(1.110)	1.406	(.597)
Delay in Return to Baseline (in seconds):	25.647	(70.874)	6.706	(12.368)	10.824	(24.156)
Frontalis EMG (in Microvolts):						
- Pretrial Baseline	4.946	(3.097)	4.672	(2.707)	3.981	(2.142)
- Trial	4.866	(2.207)	5.118	(3.738)	5.240	(2.760)
- Posttrial Baseline	4.509	(3.195)	5.024	(3.333)	4.368	(2.200)
Delay in Return to Baseline (in seconds):	18.000	(35.743)	21.529	(71.797)	14.588	(18.517)
Heartrate (in beats per minute):						
- Pretrial Baseline	70.188	(12.825)	78.335	(14.054)	73.924	(12.770)
- Trial	73.319	(13.671)	76.818	(13.677)	70.782	(10.960)
- Posttrial Baseline	72.941	(12.783)	75.465	(9.442)	74.018	(11.629)
Delay in Return to Baseline (in seconds):	10.250	(11.216)	5.765	(5.793)	8.588	(9.818)
Skin Resistance Level (in Kiloohms):						
- Pretrial Baseline	361.231	(153.240)	505.937	(212.897)	420.067	(186.512)
- Trial	341.923	(158.738)	427.625	(191.068)	351.133	(204.079)
- Posttrial Baseline	390.308	(144.624)	496.188	(212.310)	496.267	(195.583)
Delay in Return to Baseline (in seconds):	42.769	(79.308)	71.625	(97.020)	13.412	(42.195)

Means and Standard Deviations of the Physiological Variables in the PAIN Condition

	Chronic Back Pain		General Pain		Healthy Controls	
	M	(SD)	M	(SD)	M	(SD)
Left Erector Spinae EMG (in Microvolts):						
- Pretrial Baseline	3.084	(5.699)	1.279	(1.069)	1.148	(.633)
- Trial	5.284	(5.505)	1.554	(1.122)	1.404	(.624)
- Posttrial Baseline	3.674	(5.316)	1.318	(1.177)	1.241	(.823)
Delay in Return to Baseline (in seconds):	62.000	(87.002)	29.529	(71.304)	22.941	(71.523)
Right Erector Spinae EMG (in Microvolts):						
- Pretrial Baseline	1.653	(1.403)	1.772	(1.054)	1.353	(.633)
- Trial	3.950	(3.094)	2.097	(1.217)	1.575	(.566)
- Posttrial Baseline	2.643	(2.067)	1.837	(1.155)	1.502	(.787)
Delay in Return to Baseline (in seconds):	71.529	(113.500)	26.118	(70.991)	6.588	(4.403)
Frontalis EMG (in Microvolts):						
- Pretrial Baseline	4.287	(9.539)	4.912	(10.747)	4.079	(3.399)
- Trial	7.649	(6.699)	6.021	(3.110)	5.429	(3.174)
- Posttrial Baseline	4.159	(2.768)	5.404	(3.469)	4.918	(2.674)
Delay in Return to Baseline (in seconds):	30.353	(70.768)	25.412	(58.163)	13.177	(16.749)
Heartrate (in beats per minute):						
- Pretrial Baseline	71.606	(14.447)	75.247	(14.316)	73.023	(12.061)
- Trial	72.741	(14.208)	77.482	(13.492)	70.577	(11.399)
- Posttrial Baseline	72.659	(12.908)	76.753	(13.381)	73.112	(11.556)
Delay in Return to Baseline (in seconds):	12.000	(14.748)	15.765	(19.543)	5.529	(5.269)
Skin Resistance Level (in Kiloohms):						
- Pretrial Baseline	378.461	(140.745)	486.500	(199.521)	435.600	(192.904)
- Trial	303.769	(153.954)	420.562	(193.755)	361.533	(200.998)
- Posttrial Baseline	387.615	(146.738)	496.250	(195.929)	491.333	(197.361)
Delay in Return to Baseline (in seconds):	61.538	(54.609)	108.875	(111.754)	69.882	(92.894)

Means and Standard Deviations of the Physiological Variables in the MENTHAL ARITHMETIC Condition

	Chronic Back Pain		General Pain		Healthy Controls	
	M	(SD)	M	(SD)	M	(SD)
Left Erector Spinae EMG (in Microvolts):						
- Pretrial Baseline	2.929	(4.933)	1.184	(.779)	1.303	(.690)
- Trial	3.697	(5.170)	1.641	(1.204)	1.698	(.794)
- Posttrial Baseline	3.263	(5.588)	1.348	(1.113)	1.178	(.649)
Delay in Return to Baseline (in seconds):	26.647	(70.927)	49.176	(95.773)	26.000	(65.330)
Right Erector Spinae EMG (in Microvolts):						
- Pretrial Baseline	1.581	(1.158)	1.706	(1.149)	1.532	(.836)
- Trial	2.626	(1.934)	2.218	(1.621)	1.692	(.794)
- Postttrial Baseline	1.693	(1.005)	1.698	(1.079)	1.454	(.672)
Delay in Return to Baseline (in seconds):	26.941	(32.094)	11.765	(13.544)	22.118	(59.592)
Frontalis EMG (in Microvolts):						
- Pretrial Baseline	4.553	(3.618)	4.916	(2.580)	4.865	(3.721)
- Trial	5.688	(5.047)	6.046	(4.321)	5.581	(3.393)
- Posttrial Baseline	4.902	(3.496)	5.560	(3.875)	4.738	(3.091)
Delay in Return to Baseline (in seconds):	29.059	(71.281)	24.588	(71.270)	38.706	(86.174)
Heartrate (in beats per minute):						
- Pretrial Baseline	72.247	(11.874)	74.871	(10.759)	73.506	(12.403)
- Trial	73.082	(12.609)	76.735	(11.725)	75.159	(10.984)
- Posttrial Baseline	71.947	(11.513)	75.647	(10.966)	73.464	(11.651)
Delay in Return to Baseline (in seconds):	6.823	(10.345)	11.294	(12.902)	8.588	(9.919)
Skin Resistance Level (in Kiloohms):						
- Pretrial Baseline	372.846	(144.743)	494.563	(194.878)	442.333	(213.881)
- Trial	313.846	(167.897)	365.250	(167.735)	320.200	(185.998)
- Posttrial Baseline	404.077	(137.513)	459.313	(165.543)	461.867	(182.938)
Delay in Return to Baseline (in seconds):	80.923	(102.080)	150.125	(122.144)	60.941	(52.236)

Means and Standard Deviations in the Physiological Variables in the STRESS Condition

	Chronic Back Pain		General Pain		Healthy Controls	
	M	(SD)	M	(SD)	M	(SD)
Left Erector Spinae EMG (in Microvolts:						
- Pretrial Baseline	2.609	(4.802)	1.487	(1.387)	1.126	(.620)
- Trial	4.500	(5.055)	1.826	(1.488)	1.438	(.638)
- Posttrial Baseline	2.974	(4.723)	1.674	(1.398)	1.159	(.664)
Delay in Return to Baseline (in seconds):	85.294	(99.396)	65.176	(113.605)	8.118	(71.235)
Right Erector Spinae EMG (in Microvolts):						
- Pretrial Baseline	1.488	(1.603)	1.381	(.986)	1.072	(.498)
- Trial	3.597	(2.956)	1.965	(1.232)	1.581	(.599)
- Posttrial Baseline	2.163	(1.764)	1.873	(1.393)	1.338	(.584)
Delay in Return to Baseline (in seconds):	79.412	(102.944)	34.235	(76.818)	11.059	(16.173)
Frontalis EMG (in Microvolts):						
- Pretrial Baseline	3.934	(2.544)	4.750	(2.968)	4.218	(3.094)
- Trial	9.038	(6.945)	7.666	(5.840)	6.998	(4.686)
- Posttrial Baseline	5.454	(3.855)	5.988	(3.744)	5.475	(4.154)
Delay in Return to Baseline (in seconds):	49.177	(83.132)	64.118	(114.973)	53.294	(96.325)
Heartrate (in beats per minute):						
- Pretrial Baseline	73.982	(9.587)	77.829	(13.386)	74.476	(11.296)
- Trial	74.306	(11.677)	78.094	(15.956)	70.624	(11.167)
- Posttrial Baseline	71.071	(9.958)	76.871	(13.983)	73.835	(11.566)
Delay in Return to Baseline (in seconds):	5.412	(6.236)	7.259	(11.479)	6.000	(6.442)
Skin Resistance Level (in Kiloohms):						
- Pretrial Baseline	416.461	(165.630)	488.375	(193.212)	443.867	(207.246)
- Trial	309.000	(168.199)	404.563	(177.665)	339.533	(183.529)
- Posttrial Baseline	380.308	(124.995)	482.500	(193.036)	473.867	(181.819)
Delay in Return to Baseline (in seconds):	97.077	(100.192)	129.375	(129.717)	86.235	(95.512)

Baselines Values of the
Physiological and Behavioral Variables

	Chronic Back Pain		General Pain		Healthy Controls	
	M	(SD)	M	(SD)	M	(SD)
Left Erector Spinae EMG (in Microvolts):	3.626	(6.795)	1.326	(1.206)	1.226	(.633)
Right Erector Spinae EMG (in Microvolts):	2.479	(2.210)	1.564	(.980)	1.469	(.885)
Frontalis EMG (in Microvolts):	4.199	(2.713)	4.981	(2.445)	4.543	(2.469)
Heartrate (in beats per minute):	75.453	(12.478)	78.688	(15.135)	74.053	(11.996)
Skin Resistance Level (in Kiloohms):	469.286	(190.464)	639.188	(235.492)	580.667	(221.137)
Finger to Floor distance (in cm):	27.882	(25.387)	9.000	(9.906)	1.353	(3.247)

Subjective Ratings During the Experiment

	Chronic Back Pain		General Pain		Healthy Controls	
	M	(SD)	M	(SD)	M	(SD)
- PAIN -						
Pre Baseline	4.235	(2.488)	3.059	(2.561)	.118	(.485)
Post Baseline	4.765	(2.359)	3.059	(2.727)	.118	(.485)
Post Alphabet	5.529	(2.918)	3.342	(2.740)	.059	(.242)
Post Pain	5.471	(2.801)	3.529	(2.897)	.059	(.242)
Post Arithmetic	5.412	(3.063)	3.941	(3.191)	.059	(.242)
Post Stress	5.353	(2.914)	3.588	(3.142)	.529	(1.940)
- TENSION -						
Pre Baseline	3.706	(2.568)	2.294	(2.173)	2.118	(1.453)
Post Baseline	4.588	(2.671)	2.941	(2.904)	1.706	(1.896)
Post Alphabet	5.588	(3.001)	3.471	(2.125)	2.118	(1.799)
Post Pain	4.765	(2.927)	3.823	(2.456)	1.529	(1.463)
Post Arithmetic	5.000	(2.979)	4.235	(2.818)	3.176	(2.721)
Post Stress	5.412	(2.980)	3.823	(2.230)	2.706	(2.312)
- UNPLEASANTNESS -						
Pre Baseline	4.412	(2.647)	2.529	(2.503)	1.941	(1.435)
Post Baseline	4.529	(3.023)	3.529	(3.064)	2.294	(2.144)
Post Alphabet	2.353	(2.644)	1.941	(2.135)	.588	(.870)
Post Pain	3.765	(3.667)	4.647	(3.297)	2.471	(1.972)
Post Arithmetic	4.765	(1.113)	5.235	(3.212)	4.118	(3.295)
Post Stress	5.353	(2.234)	4.824	(3.147)	4.118	(2.870)
- EFFORT -						
Pre Baseline	4.235	(3.580)	2.000	(2.208)	1.588	(1.770)
Post Baseline	5.353	(3.656)	2.823	(2.675)	2.235	(2.611)
Post Alphabet	1.941	(1.886)	1.882	(2.288)	.294	(.588)
Post Pain	3.529	(2.939)	3.000	(2.291)	2.177	(1.912)
Post Arithmetic	5.647	(2.548)	6.059	(3.631)	5.412	(3.318)
Post Stress	4.000	(2.318)	3.118	(2.421)	4.059	(2.609)

Means and Standard Deviations of the Subjective Variables in the Three Groups

	Chronic Back Pain		General Pain		Healthy Controls	
	M	(SD)	M	(SD)	M	(SD)
STAI-S Pre Session	41.000	(9.104)	41.824	(9.838)	33.176	(6.337)
STAI-S Post Session	43.706	(9.198)	41.294	(10.005)	30.647	(6.461)
Pain Card Index	2.953	(1.012)	2.150	(1.026)	t(17) = 2.09*	
McGill Word Group	1.188	(.544)	1.357	(.497)	t(28) = - .89	
McGill Present Pain Intensity	2.375	(.957)	2.214	(.699)	t(30) = .48	
McGill Affective Dimension	.289	(.199)	.204	(.248)	t(30) = 1.07	
McGill Evaluative Dimension	.611	(.284)	.450	(.278)	t(30) = 1.61	
McGill Other Dimension	.426	(.227)	.334	(.195)	t(30) = 1.24	
McGill Total Score	.427	(.199)	.343	(.194)	t(29) = 1.20	
Depression Adjective Checklist						
DACL Negative Adjectives	4.177	(1.187)	5.769	(4.512)	t(28) = -1.00	
DACL Positive Adjectives	2.235	(1.522)	1.308	(1.888)	t(28) = 1.49	
DACL Total Adjectives	12.353	(4.609)	15.615	(5.824)	t(28) = -1.70	
Back Depression Inventory	10.500	(6.044)	17.600	(9.425)	t(29) = -2.48*	
Health Locus of Control (HLC)						
HLC (internal)	23.941	(4.854)	23.188	(3.468)	t(31) = .51	
HLC (powerful other)	18.706	(8.752)	20.500	(6.603)	t(31) = - .66	
HLC (chance)	17.353	(8.276)	17.813	(5.624)	t(29) = - .19	
STAI-S	42.941	(9.596)	48.786	(14.192)	t(29) = -1.36	
Locke Wallace Marital Adjustment Scale	114.833	(15.165)	91.750	(24.944)	t(18) = 2.59*	
Pain Experience Questionnaire	122.917	(32.047)	133.727	(22.059)	t(21) = - .93	

* $p < .05$

LEBENSLAUF

Ich wurde am 23.4.1954 als ältestes Kind des Landwirtsehepaars Maria und Georg Flor in Schnaittenbach geboren. Nach dem Besuch der Volksschule Schnaittenbach von 196o bis 1965 trat ich in das Gymnasium der Armen Schulschwestern v. U.L.F. in Amberg über. Dort besuchte ich den Neusprachlichen Zweig von 1965 bis 1971 und 1972 bis 1974 und legte 1974 die Reifeprüfung ab. Das Schuljahr 1971/72 verbrachte ich an der Pequea Valley High School in Kinzer, Pa.,USA, und erwarb dort 1972 das High School Diploma.

An der Universität München belegte ich im Winstersemester 1974/75 Amerikanistik, Politikwissenschaft und Publizistik. Das Studium der Psychologie begann ich im Sommersemester 1975 an der Universität Würzburg, und im November 1977 legte ich mein Vordiplom in Psychologie ab. Vom Wintersemester 1977 bis zum Sommersemester 1981 setzte ich mein Psychologiestudium an der Universität Tübingen fort, wo ich im September 1981 das Diplom-Zeugnis erhielt.

Von September 1981 bis September 1982 studierte ich als DAAD-Stipendiatin, von September 1982 bis Februar 1983 als Special Student, an der Yale University in New Haven, Ct., USA. Gleichzeitig arbeitete ich als wissenschaftliche Mitarbeiterin und Therapeutin an der Schmerzklinik des Veterans Administration Medical Centers in West Haven, Ct., USA, und als Therapeutin an der Yale Behavioral Medicine Clinic in New Haven, Ct., USA. Seit Mai 1983 bin ich als wissenschaftliche Hilfskraft am Psychologischen Institut der Universität Tübingen, Abteilung Klinische und Physiologische Psychologie, beschäftigt.